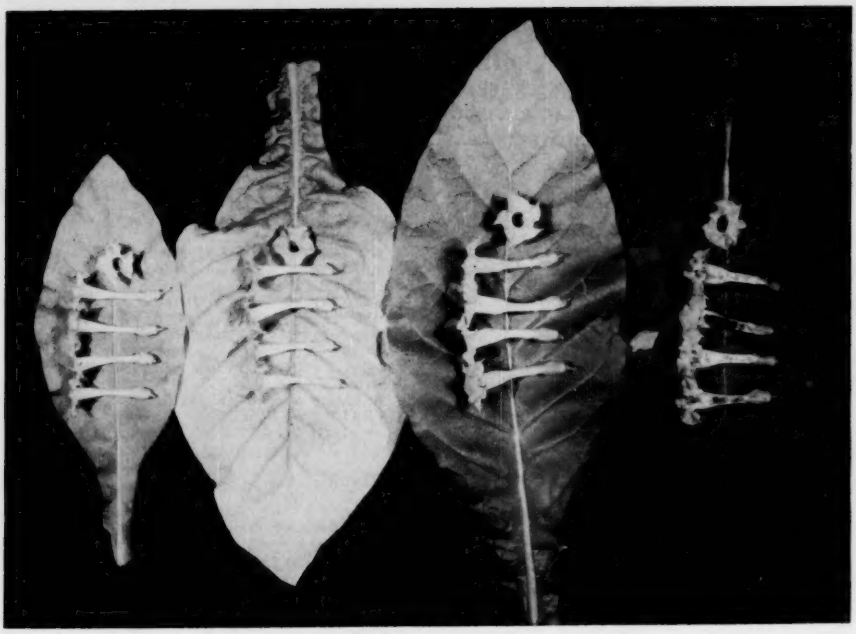
The Journal of HEREDITY



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Nectarless Muskmelons

Taillessness in Manx Cats

Opossum and Spastic Mice

Mormoniella Eye-Color Mutants

World Distribution of Cattle Types

VOLUME 52 SEPTEMBER-OCTOBER, 1961 NUMBER 5

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CHROMOSOMES FROM A GREEN PLANT

Figure 1

Shown above are somatic chromosomes from a green plant of the ever-sporting type of *M. incana* identified according to chromosome pair; shown below are chromosomes from the above cell arranged to illustrate the heteromorphic pair 6 and 6′ in the green plants.

SOMATIC CHROMOSOMES IDENTIFIED IN THE EVER-SPORTING TYPE OF STOCK

J. G. Ross and W. H. Miller*

THE ever-sporting single-flowered form in stock, *Matthiola incana* (L.) R. Br., was investigated in a survey to find material heterozygous for chromosome markers which could be used to test the hypothesis of somatic reduction, advanced to explain the homozygous nature of mutants appearing after colchicine treatment of sorghum[2,8]. The ever-sporting mechanism, symbolized *l S/+ s* by Johnson[4,5], is explained by Frost[3] as due to the close linkage on one chromosome of pollen lethal *l* with the gene *S* for single flowers, while its homologue carries the factor for viability, +, and *s* for double flowers. Johnson[4,5] has studied the effect of crossing-over between these genes. Closely linked to *s* is gene *v*, causing virescence, which is clearly expressed in homozygous seedlings treated with temperatures below 60° F. (Johnson, unpublished).

Disregarding the possible effect of crossing-over, the identifiable green seedlings would have the genetic constitution *l S V/+ s v* and therefore should provide an excellent opportunity for testing the hypothesis. Philp and Huskins[7] observed the absence of a satellite in the ever-sporting single-flowered form and suggested that this might be the cause of the pollen lethal. A study of the somatic chromosomes accordingly was made to determine if the lethal *l* might be recognized cytologically to afford a cytological as well as a genetic marker.

Materials and Methods

A columnar (single-stemmed) line of the ever-sporting stock was obtained from Dr. B. L. Johnson, Floriculture Department, U.C.L.A. It contains *v* for virescence closely linked to the double-flowered gene *s*. The double flowers have neither stamens nor pistils so seed is produced only on the singles. Plants from this seed segregate 46 percent single: 54 percent double-flowered in each successive generation because of the effect of the lethal *l* on the pollen

TABLE I. Comparison by the t-test of means of the ratios of the short to long arm (S.A./L.A.) of the shorter with the longer chromosome within each homologous pair in green (17 cells) and virescent (16 cells) plants

	Green plants Chromosome length			Virescent plants Chromosome length		
Chromosome no.	Short S.A./L.A.	Long S.A./L.A.	Difference	Short S.A./L.A.	Long S.A./L.A.	Difference
1	.549	.546	.003	.554	.546	.008
2	.547	.540	.007	.532	.530	.002
3	.498	.516	.018	.521	.517	.004
4	.543	.538	.005	.546	.565	.019
5	.905	.892	.013	.916	.921	.005
6	.761	.874	.113*	.872	.876	.004
7	.820	.823	.003	.813	.811	.002

*P<.01 (t-test, paired variates)

*Agronomy Department, South Dakota State College. Approved for publication by the Director of the South Dakota Agricultural Experiment Station as Journal Series No. 497. The senior author acknowledges the use of the facilities and the many kindnesses of the staff of the Institute of Genetics, Lund, Sweden, while on a Guggenheim Fellowship when the original observations were made. This investigation was supported in part by research grant G1672 from the National Science Foundation.

and, to a small degree, on the ovules or zygotes (Johnson[6]). The gene *v* makes identification of the virescent doubles as opposed to the green heterozygous seedlings possible at the early seedling stage if they are grown at approximately 60° F.

Root tips for squash preparations obtained from plants identified when seedlings as green (*Vv*) or virescent (*vv*) were placed for seven hours at 3 to 5° C. in .1 percent aqueous solution of colchicine, after which they were fixed in 3:1 alcohol-acetic for 15 to 24 hours and stained with aceto-orcein (synthetic) as described by Tjio and Puck[9]. The measurements were made with an ocular screw micrometer (Bausch and Lomb) in a manner similar to that described by Blixt[1]. In order to minimize errors, three measurements were taken on each arm of every chromosome and the mean recorded. Seventeen cells in green plants and 16 in virescent were selected for excellent spreading of the chromosomes and straightness of their arms so that precision in measurement might be obtained.

Results

During examination of the chromosomes in cells of the green plants, a single chromosome was observed which had a slightly submedian centromere and approached in size the longest chromosome within the cell. Such a cell is shown in the top half of Figure 1, which illustrates the slightly submedian chromosomes 6 and 6′, the shorter member of this heteromorphic pair with centromere located farther from the median position. The seven pairs are arranged in increasing length in the bottom half of Figure 1, and each pair is given a number in accordance with its rank in length. In pair 6 the difference in length and morphology of the two chromosomes clearly illustrates that a deficiency in the short arm has occurred. Such a difference was not found in Figure 2 (top), where chromosomes from a virescent plant are shown. Both chromosomes 6 were found to be long and of slightly submedian type, as represented by 6 in the green plants. The seven pairs from the virescent are shown in Figure 2 (bottom); no heteromorphic pair was found. Since differences in contraction could perhaps occur so that the appearance of the heteromorphic pair might be more apparent than real, measurements of each arm of each chromosome were made in the best cells (17 in the green and 16 in the virescent) that could be located. The ratio of the short to long arm was computed for each member of each pair. The means of these ratios for each of the shorter and longer of each pair for green and virescent plants are shown in Table I. It will be noted that no significant difference, using the t-test for paired variates, was found between the ratios, except for chromosome pair 6 in the green plants in which a highly significant difference occurred. These measurements verify the observation that a heteromorphic pair occurs in the green but is absent in the virescent plants.

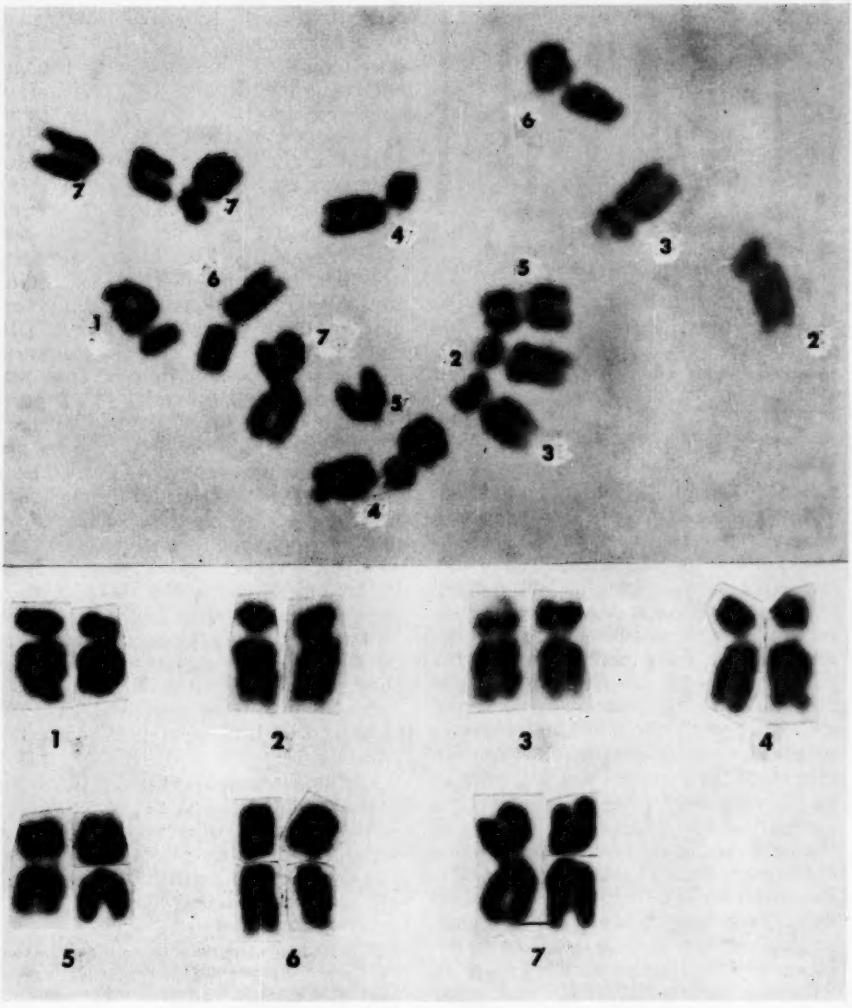
Although a difference between the

TABLE II. Comparison by the t-test of means of the ratios of the short to long arm (S.A./L.A.) for each homologous pair of chromosomes in green (17 cells) with those in virescent (16 cells) plants

Chromosome no.	Green plants		Virescent plants		
	N	S.A./L.A.	N	S.A./L.A.	Difference
1	34	.548	32	.550	.002
2	34	.544	32	.531	.013
3	34	.507	32	.519	.012
4	34	.540	32	.555	.015
5	34	.898	32	.919	.021
6	17	.874(long)	32	.874	.000
6′	17	.761(short)			.113†
7	34	.821	32	.812	.009

*P<.01 (t-test)

†Difference based on 6 in the virescent plants.

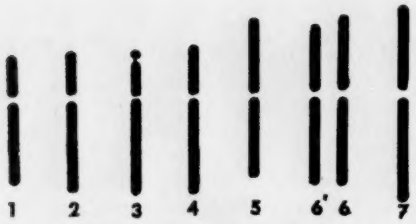


CHROMOSOMES FROM A VIRESCENT PLANT

Figure 2

Shown above are somatic chromosomes from a virescent plant of the ever-sporting type of *M. incana* identified according to chromosome pair; shown below are chromosomes from the above cell arranged to illustrate the absence of a heteromorphic pair in the virescent plants.

green and virescent plants in respect to the relative contraction of the arms for the different chromosomes would not be expected, it was desirable to determine the significance of any difference in the ratios of the arms to rule out the possibility of deficiencies involving other chromosomes. The significance of these differences was tested by means of the t-test for independent variates,



IDEOGRAM OF *M. INCANA* CHROMOSOMES

Figure 3

Ideogram representing the chromosomes of the ever-sporting type of *M. incana*. Sixty-six measurements are represented by each diagram except for 6′ (17 measurements) and 6 (49 measurements).

as shown in Table II where the mean ratios of the short to long arms for each of the chromosome pairs for green and virescent plants are given. No significant difference was found except between 6 and 6′, as would be expected from previous tests. Measurements for each of the chromosome pairs may therefore be combined to obtain the relative sizes of the chromosomes, as in Table III where, except for chromosomes 6 and 6′, 66 individual chromosomes have been used in computation of each of the means. Data from the 49 normal No. 6 chromosomes have been included in the mean for 6, while data from the 17 No. 6′ chromosomes found in the green plants have been included in the mean for 6′. From these measurements an ideogram of the chromosomes of this ever-sporting line of *Matthiola incana* has been constructed (Figure 3). The standard deviation for each chromosome indicates the variation in contraction that might be expected between cells after colchicine treatment. In general, contraction was uniform, but in order to be certain of the presence of the heteromorphic pair it was necessary to make a number of measurements in different cells.

The numbering of the chromosome pairs in order of increasing size forms a basis for future cytogenetic work on this species. Chromosome 3 can be recognized as the satellite chromosome, while chromosomes 1, 5, 6 and 7 can be identified with assurance by their characteristic length and the position of their centromeres. Chromosomes 2 and 4, however, are difficult to separate except on the basis of a small difference in length. Assortment into their respective pairs can nevertheless be accomplished with a fair degree of confidence.

The lethal *l*, which causes complete non-function of *l* pollen and, according to Johnson[6], a calculated viability coefficient of .83 for eggs, appears to be caused by a deficiency in the short arm of chromosome 6, the chromosome designated 6′ in this study. The appearance of 6′ in the green plants, which are the ever-sporting singles, and its absence in the virescent plants which bear double flowers would seem conclusive proof for the segregation of this chromosome coincident with the lethal. This observation agrees with that of Philp and Huskins that a deficiency might be involved in causing a pollen lethal, but the location of the deficiency does not involve a missing satellite as they observed. Both satellited chromosomes, pair 3, were seen in all cells observed in both the green and virescent plants.

Chromosome 6 would, therefore, carry the lethal *l* and the genes *s* and *v*. Johnson[6] reports other recessives affecting chlorophyll or plastid production, flower color and plant habit as belonging in the same linkage group.

TABLE III. Mean lengths of 66 chromosomes from the combined green and virescent plants (except for the heteromorphic pair found in the green plants) representing each of the seven pairs in *Matthiola incana*. The standard deviation illustrates the degree of contraction between cells

Chromosome no.	N	Short arm μ	Long arm μ	Total μ
1	66	1.32	2.41	3.73±.33
2	66	1.40	2.63	4.03±.65
3	66	1.41	2.76	4.17±.75
4	66	1.53	2.82	4.35±.76
5	66	2.22	2.42	4.64±.87
6	49	2.29	2.63	4.92±.83
6′	17	2.06	2.70	4.76±.71
7	66	2.54	3.11	5.65±.90

This recognizable deficiency in chromosome 6 should provide a valuable tool for testing the occurrence of somatic reduction after colchicine treatment of heterozygous seedlings, especially since it occurs in a columnar line. Since in this line no latent buds other than the primary epicotyl are present at the time of treatment, any new growth which arises would be from nuclei which have undergone influences within the colchicine-induced tumor.

Summary

Chromosomes of an ever-sporting columnar line of stock were examined. Plants heterozygous for a pollen lethal were found to have a heteromorphic pair. Homozygous plants did not contain the shorter member of this pair. Measurements corroborated this observation and indicated that the pollen lethal *l* was caused by a deficiency on chromosome 6, so designated when the chromosome pairs were arranged according to increasing length. Further cytogenetic study of this species should be facilitated by these observations. The genetic and cytological chromosome markers in this line should prove useful in making tests for the occurrence of somatic reduction in colchicine-induced tumors.

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Correction—Inheritance of Dieldrin Resistance in the Housefly

Two errors appeared in the recent paper by Mohammad Abdullah, "Inheritance of Dieldrin Resistance in the Housefly" (*Jour. Hered.* 52: 179–182. 1961). On page 179 under Materials and Methods, the first sentence in column two should read "... an F_1 generation was obtained, a portion of which was allowed to inbreed to produce an F_2 generation" *instead of* "... backcrossed with the susceptible strain." In Tables II and III *read* R *instead of* F_1 under Type, (Original Cross).

The editors regret the errors.

ANOTHER ALLELE AT THE W LOCUS OF THE MOUSE

JACK BALLANTYNE, FRED G. BOCK, LEONELL C. STRONG AND WALTER C. QUEVEDO, JR.*

RECENTLY there appeared in our colony of C57/St mice a new mutant characterized by dominant white spotting similar to that caused by alleles of *W*. Mutations of the *W* locus have been reported by a number of investigators[2,4,5], and it has been suggested by Russell *et al.*[4] that the locus appears to be relatively mutable. A similar suggestion was made in 1943 by Grüneberg[1] but was not repeated in a later edition[2] of the same work. It is of interest that no new mutations were reported between the dates when the two editions were prepared. The purpose of this paper is to describe the new mutant and to present evidence that it represents, indeed, a new allele of *W*.

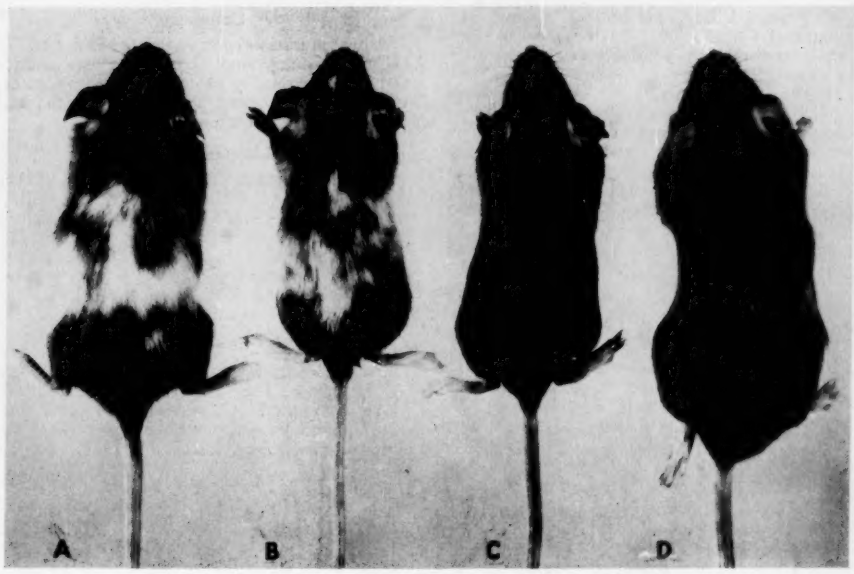
A spotted female appeared in a litter of C57/St mice which had been maintained in a subline by brother-to-sister matings. The animal showed an extensive diffuse-white-spotting of the back and sides with a small frontal blaze. The ventrum was largely white. Pigmented areas on the dorsum were appreciably lighter in color than the intense black characteristic of C57/St mice. Microscopic examination of hairs plucked from these regions revealed a variation in pigmentation of individual hairs which permitted their classification as follows: (1) totally white, (2) pigmented at tips but non-pigmented for variable lengths basally, (3) pigmented at tips and irregularly pigmented throughout length, and (4) relatively uniformly pigmented from tip to base. The "basal dilution" of pigmentation observed in many hairs appeared to be associated with a striking reduction in medullary pigmentation. Matings between the mutant and a pedigreed C57/St male revealed a dominant pattern of inheritance. Subsequent matings were conducted between spotted offspring of the original female, giving rise to 123 young. Of these, 34 (28 percent) were black, 56 (46 percent) were spotted, and 33 (27 percent) appeared pale at birth and were presumed to be anemic. The median life span of the "anemic" mice was eight days (Table I), and the pale mice that lived developed into sterile black-eyed whites. These features are typical of those caused by various alleles at the *W* locus[2].

Tests were made by mating the mutants to mice bearing $W^v w$ on a C57BL background. Of 39 young, 12 (31 percent) were black, 20 (51 percent) were spotted, and seven (18 percent) were pale (Table II). The spotted mice appeared to fall into two classes (Figure 4). Five of the seven pale mice lived to become black-eyed white adults. Matings between the mutant and mice bearing an allele of *W* described by Strong and Hollander[5] also provided anemic young which grew to be black-eyed whites. Inasmuch as the tests supported the belief that the mutation occurred at the *W* locus, the symbol W^b is proposed for the new mutant and W^s for the allele reported by Strong and Hollander. This nomenclature is in accordance with the practice initiated by Russell *et al.*[4]

TABLE I. Longevity of W^bW^b mice

Days lived	Number of W^bW^b mice
0	—
1	7
2	3
3	2
4	2
5	1
8	2
21	1
>50	15
	Total 33

*Biological Station, Roswell Park Memorial Institute, Springville, New York. The authors wish to express their appreciation to Dr. Elizabeth Russell of the Roscoe B. Jackson Memorial Laboratory who furnished the $W^v w$ mice used in these tests.



DIFFERENTIAL CHARACTERISTICS OF MICE BEARING W^b

Figure 4

A—W^bw (dam); *B* and *C*—F_1 offspring; *D*—W^vw (sire). W^bW^v (black-eyed white) and *ww* (self) are not illustrated. The difference in spotting patterns is evident. The greater lightening of the pigmented areas of W^bw mice is not so apparent in the photograph.

Differential Characteristics of the Mutant

When the mutant appeared, it was thought important to rule out the possibility of a vicarious mating. W^v had been maintained in this area for six years prior to the appearance of the new mutant. The gene was maintained in an "ultimate dominant" stock housed in a building separate from that in which the mutation occurred. The "ultimate dominant" stock is homozygous for the genes for wavy, agouti, pintail, loop, and also piebald, in addition to being heterozygous for W^v. None of these traits characteristic of the "ultimate dominant" stock were observed in the original mutant, her siblings, or in any of her offspring. Further, when her W^b offspring were backcrossed to pedigreed C57/St mice for two generations, no change in the expression of W^b was noted.

While it seemed extremely unlikely that the mutant was the result of a mating between the C57/St mother and a wild mouse bearing the W^v gene, specific differences between W^v and W^b were necessary to support the contention that a mutation had occurred. For this purpose, the "ultimate dominant" stock was backcrossed to C57/St for three and four generations. The following differences between the two lines persisted in spite of the relative similarity in genetic background: (1) W^b is characterized by extensive spotting with a frontal blaze; W^v causes only occasional spotting of the back with no blaze. (2) W^b leads to a pronounced decrease in pigment granules in the base of the shaft of colored hairs; only a very slight effect was noted in W^v mice. (3) W^bW^b mice were born alive, and many lived to maturity; in 74 young born to the W^v stock, only two anemic young were observed, and these died within three days of birth.

Of the alleles now recorded at the *W* locus, W^b most nearly resembles W^v in its expression[3]. However, W^b mice are characteristically more extensively spotted than those genotypically

TABLE II. Allelism between W^b and W^v

Litter number	Number born	Black	Like W^v	Like W^b	Black-eyed white or lethal "anemic"
1	8	2	3	1	2
2	8	3	—	3	2
3	7	3	1	3	—
4	7	2	3	2	—
5	5	—	1	1	3
6	4	2	1	1	—
Totals	39	12	9	11	7

W^v (Figure 4) and microscopic examination of pigmented hairs from W^v mice failed to reveal the extensive terminal deficiency of medullary pigmentation regularly observed in W^b hairs. In hair samples from W^v mice, occasional totally white hairs were observed, and the pigmented hairs appeared less intensively darkened than those from corresponding *ww* mice[3]. However, W^b mice were characterized by marked irregularity of medullary pigmentation and total absence of basal pigmentation in many hairs with pigmented tips.

It is noteworthy that this mutant represents the second mutation at the *W* locus observed in this laboratory. At the present time, five different alleles of *W*, in addition to the wild type, are known: *W*, W^v, W^s, W^i, and W^b, each giving somewhat different effects in spotting or degrees of anemia. More than one mutation to several of the alleles has been reported[4]. The diversity of alleles and the frequency with which mutations are observed lend support to the suggestions that the locus is unstable.

Summary

A new mutation occurred at the *W* locus. On a C57/St background, the mutant was characterized by extensive spotting in heterozygotes, whereas homozygotes, apparently anemic, had a median life span of eight days.

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A PINK-EYED DILUTION IN THE CAT

IT WAS the author's original intention to report in detail on the genetic basis, if any, for the cat to be described below. However, her untimely death due to an intestinal obstruction ended the work almost before it was started.

During the summer of 1958 three kittens from a litter were turned over to the S.P.C.A. in Springfield, Massachusetts. Their estimated date of birth was April, 1958. Two of these animals were apparently quite ordinary, although no detailed description of them was available. The mother was presumably normal; the father was unknown. The third kitten, a female, had pink eyes (although a small amount of yellow pigment was present in the iris) and a light tan coat. She was a blotched tabby (dominant) with moderate white spotting (dominant) and had a slight kink at the tip of her tail. Her hair was short and dense with a fine texture. In all other respects she conformed to the wild type. It is suspected that she had some visual impairment from the rather clumsy way she would run into things and from her hesitancy to jump from a height which would not disturb most cats.

The animal apparently never came into heat spontaneously during the first three years of her life, but this may have been due to her caged existence rather than to any inherited or congenital defect. She was treated with hormones in January, 1961, and subsequently mated to a chocolate point Siamese. This cross was made with the purpose of discovering if any relationship existed between her phenotype and the albino series of alleles. A chocolate point Siamese cat is homozygous recessive for a pigment-determining allele at the albino locus (c^bc^b), for brown (*bb*) and for non-tabby (*aa*).

The cross produced a litter of three kittens, born 10 days prematurely with the result that none survived. However, all were fully pigmented tabbies and therefore of the genotype $+^c c^b +^b b +^a a$. From the data it can be concluded that the phenotype of the mother was due (if indeed genetically determined) to the homozygous recessive condition at some locus other than that of the albino series or the black-brown alternatives.

NEIL B. TODD

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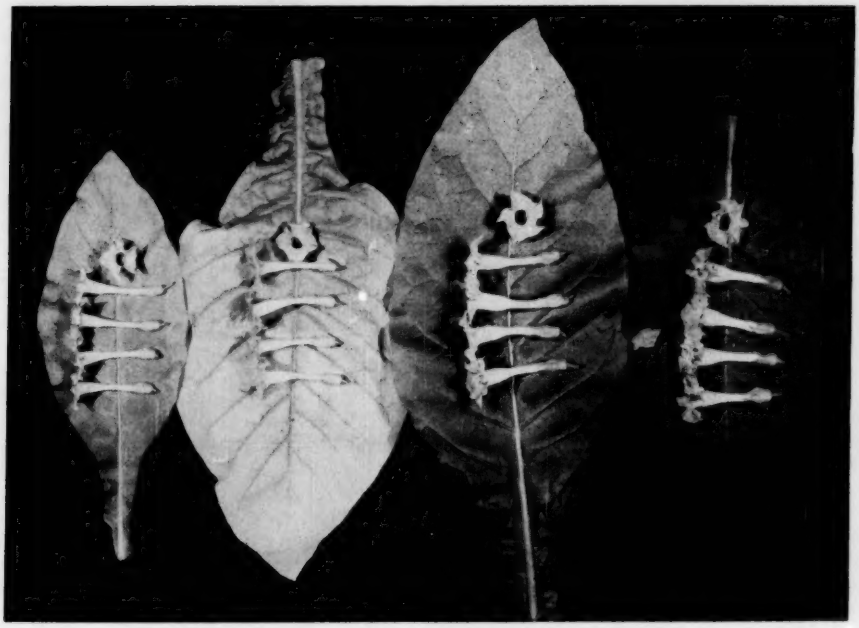
Harvard University

HAPLOID NICOTIANA DEFICIENT FOR TWO CHROMOSOMES

L. G. Burk and D. U. Gerstel*



FLORETS AND LEAVES OF YELLOW-GREEN PLANTS

Figure 5

Left to right—florets, on leaves of comparable maturity, from sub-haploid, haploid, diploid and autotetraploid yellow-green plants.

COLCHICINE is the most widely used agent for doubling the chromosome complements of plants. Franzke and Ross[2,3] have reported that colchicine may also produce homozygous mutations in sorghum. Smith[4] has pointed out that colchicine can induce loss of chromosomes in *Nicotiana*, since he obtained a range of chromosome numbers from haploid through aneuploid to higher polyploid levels. It has also been observed that polyploid and aneuploid plants appear following treatment with colchicine during the early seedling stage. A few ostensibly diploid plants with altered phenotypes were also found in the treated populations.

A study was undertaken to determine the origin of new phenotypes, and for this purpose a heterozygous population was produced by crossing normal green (*Yg Yg*) with yellow-green (*yg yg*) *N. tabacum* plants. The *yg* allele, which served as a seedling marker, is the same

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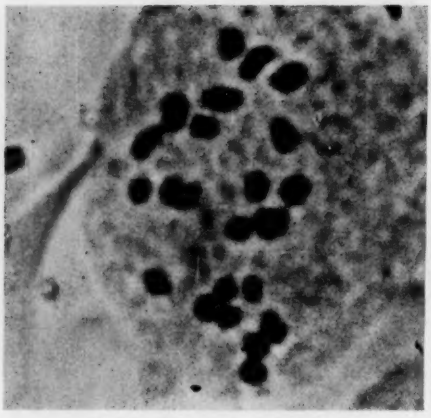


MEIOTIC METAPHASE I

Figure 6

A meiotic cell at first metaphase containing 22 univalents.

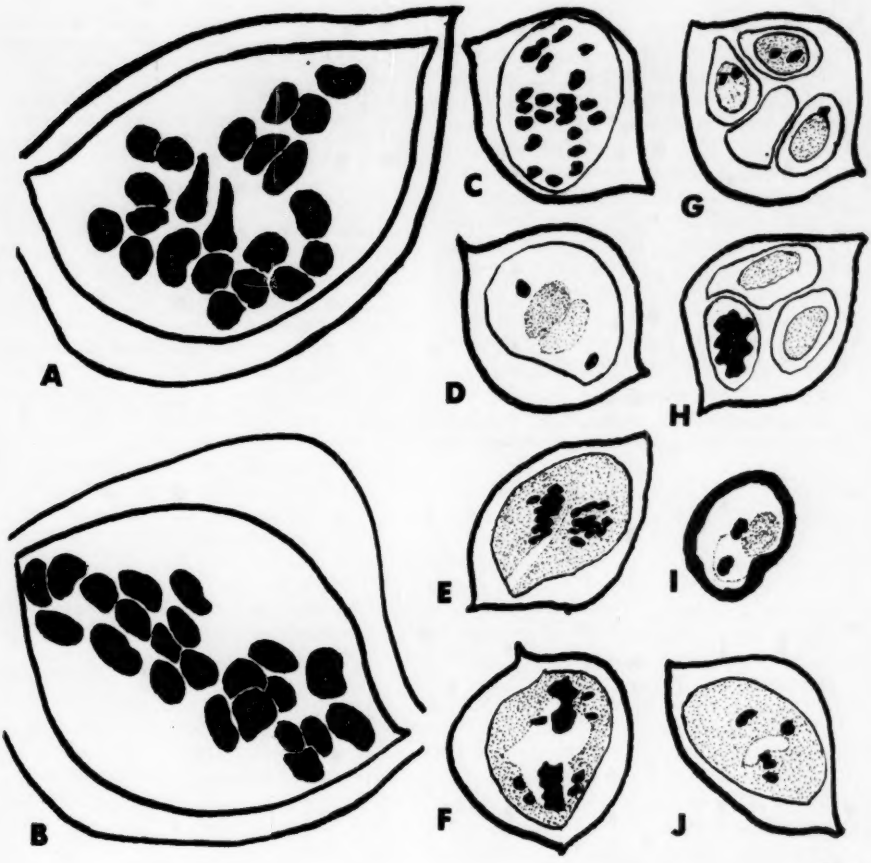
as the one called the "Consolation" factor, after the variety in which it was first discovered. The choice of a heterozygote carrying a marker gene was dictated by a need to distinguish contaminants from any possible mutations induced by colchicine. Thus, an F_1 hybrid with a new phenotype could be checked by the segregation of F_2 progeny into green and yellow-green classes.

This report is concerned with a single yellow-green plant detected at the cotyledon stage among more than 5000 seedlings of an F_1 (*Yg yg*) population treated with colchicine. The plant grew quite slowly and produced flowers when it had reached a height of approximately 14 inches. Judged on the basis of stainability, pollen fertility was negligible. However, when pollinated by normal *Yg Yg* or *yg yg* plants, the dwarf set an average of three seeds per capsule. Figure 5 illustrates leaves and florets of yellow-green plants with 22, 24, 48 and 96 chromosomes.

The following cytological observations were made. Meiotic metaphase I plates usually contained 22 univalent chromosomes (Figure 6), but an occasional cell showed fewer chromosomes. The dispersion of chromosomes at metaphase and the lack of a precise anaphase, together with irregularities in microspore development pictured in Figure 7, are characteristics of haploids or interspecific hybrids between distantly related species of *Nicotiana*. Such wide species hybrids are, in effect, amphihaploids. Another rather characteristic feature of haploidy was the occurrence of a wide variety of meiotic stages in cells from a single anther. In mitotic preparations from young leaves and root tips, 22 chromosomes were observed, except in one preparation from leaf tissue which contained cells with both 18 and 22 chromosomes. To the authors' knowledge this plant represents the first reported occurrence of a viable tobacco plant with less than the haploid number of chromosomes; nor are they aware of any reports of nullisomics in diploid ($2n = 24$) tobacco.

The method employed in this study of treating germinating tobacco seed with colchicine differed slightly from Smith's[4] technique. Colchicine at a concentration of 0.4 percent was applied for two to four hours to germinating seedlings—a briefer period than Smith had used. After treatment the seedlings were suspended in water and then sprinkled on the surface of soil-filled pots in the manner of dispensing salt and pepper. Smith had discarded the fastest growing seedlings, but in this experiment those which grew slowly were discarded. The latter were undesirable because they represented predominantly heteroploid and polyploid forms. Fertility in such plants is often low, and therefore opportunities for identifying genetic changes are minimized. As a consequence of the limited time of treatment and the retention of early emerging seedlings, many of the plants were unaffected diploids.

For a number of reasons, natural causes, rather than the effect of colchicine, were probably responsible for the origin of the sub-haploid plant. Colchicine has produced sectorial chimeras in some material observed by the authors, and therefore its action could have been responsible for the presence of 18 rather than 22 chromosomes in some cells,



IRREGULARITIES IN MICROSPORE DEVELOPMENT

Figure 7

Camera lucida drawings of stages in microsporogenesis.

although spontaneous variation of chromosome numbers in somatic tissues has been reported in numerous plants and animals. However, it is difficult to credit colchicine with the almost universal occurrence of 22 chromosomes throughout the plant. Coupled with this is the yellow-green appearance of the cotyledons. Since cotyledons are fully laid down in the mature seed, their phenotype should not be altered by colchicine treatment during germination.

If colchicine were assumed to be the cause of development of the sub-haploid plant, certain improbabilities would have to be accepted. First, the seedling was originally diploid and colchicine brought about the deletion of 26 chromosomes. Second, simultaneous deletion occurred in tissues of root and shoot. Third, the seedling was haploid and colchicine caused the simultaneous deletion of two chromosomes in both primordia. These views are unlikely.

More probably, the sub-haploid plant originated androgenically, since the male, but not the female parent, carried alleles for yellow-green. Androgenic haploids of *Nicotiana* have been reported by Clausen and Lammerts[1] and by others. The two deleted chromosomes which placed the plant in a sub-haploid rather than a 24-chromosome haploid condition may have been lost at an early stage in development. However, the manner in which the chromosomes were lost is, perhaps, less important than the ability of the plant to grow and flower with two chromosomes less than a complete haploid set.

Summary

A sub-haploid *N. tabacum* plant with 22 chromosomes was isolated from an *intra*specific population treated with colchicine. However, colchicine, which is known to cause duplication and deletion of chromosomes, was probably not responsible for the development of the unusual plant. It is suggested that the plant was of androgenic origin and that two chromosomes were lost from the haploid complement at an early stage in development. Some leaf cells contained only 18 chromosomes.

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WORLD DISTRIBUTION OF THE MAJOR TYPES OF CATTLE

RALPH W. PHILLIPS*

IN THE five to six thousand years since cattle were domesticated on the hilly, grassy, open-forested flanks of the Near Eastern mountain ranges[10], cattle have spread over the globe in the service of man. And, either under man's guiding hand or by accident of nature, a host of types and breeds has emerged. Among these types and breeds, some have found their place in the lush pastures of the well-watered portions of the temperate zones; others have adjusted to the hot, humid weather and the coarse feed of the tropics; still others serve man in the cold highlands and well into the northern and southern latitudes. Some are specialized for dairy production, others for beef. The dual-purpose animal has also found its place for the combined production of beef and milk. In many areas, triple-purpose animals have been the primary type, providing man with milk, meat and draft power. In still other areas, where religious taboos prohibit the eating of beef, the emphasis has been on animals for draft or for milk production or for a combination of these two functions.

This host of local types and breeds may be classified broadly into two major groups or types, the humpless cattle of European and northern Asiatic origin (*Bos taurus*), and the humped Zebu cattle of southern Asiatic origin (*Bos indicus*). The object of this paper is to indicate the manner in which cattle belonging to these major types, and to types intermediate to them, are distributed around the world.

Something of the complexity of the breeds and types of cattle now in existence is indicated by the map in Figure 8, showing the approximate points of origin of the major breeds and many of the minor breeds of cattle. The Zebu breeds listed in India and Pakistan are those described by Joshi and Phillips[3]. The African types and breeds are those described by Joshi *et al.*[2] Types shown as originating in China and Mongolia are described by Phillips *et al.*[9] Breeds originating in the U.S.S.R. are those described by Novikov *et al.*[7] For the most part, the other breeds shown on the map are those listed for the respective countries by Mason[5].

Among the 195 breeds and types shown on the map, there are no doubt some which may not qualify as distinct breeds. On the other hand, the listings are not all-inclusive and some have been omitted which might well qualify as breeds. For example, only eight breeds are shown as originating in Italy, yet a recent publication[1] describes 23 breeds and local types that are native to that country. Also, in several instances, for example, in Portugal and Spain, a number of more or less distinct breeds are covered by a single number. This map is, therefore, not presented as a means of indicating all the recognized breeds and local types of cattle, but rather as a means of emphasizing the great number of types and breeds of cattle that do exist.

The information available in the publications already mentioned, and from other sources, was used in compiling the map in Figure 9, which shows the approximate distribution of the two basic types and the cattle intermediate to these types.

In such a generalized map, only the prevailing type in each area can be shown. Even this is difficult, particularly for areas such as those in which Zebu cattle are shown as having been recently introduced, *i.e.*, within the

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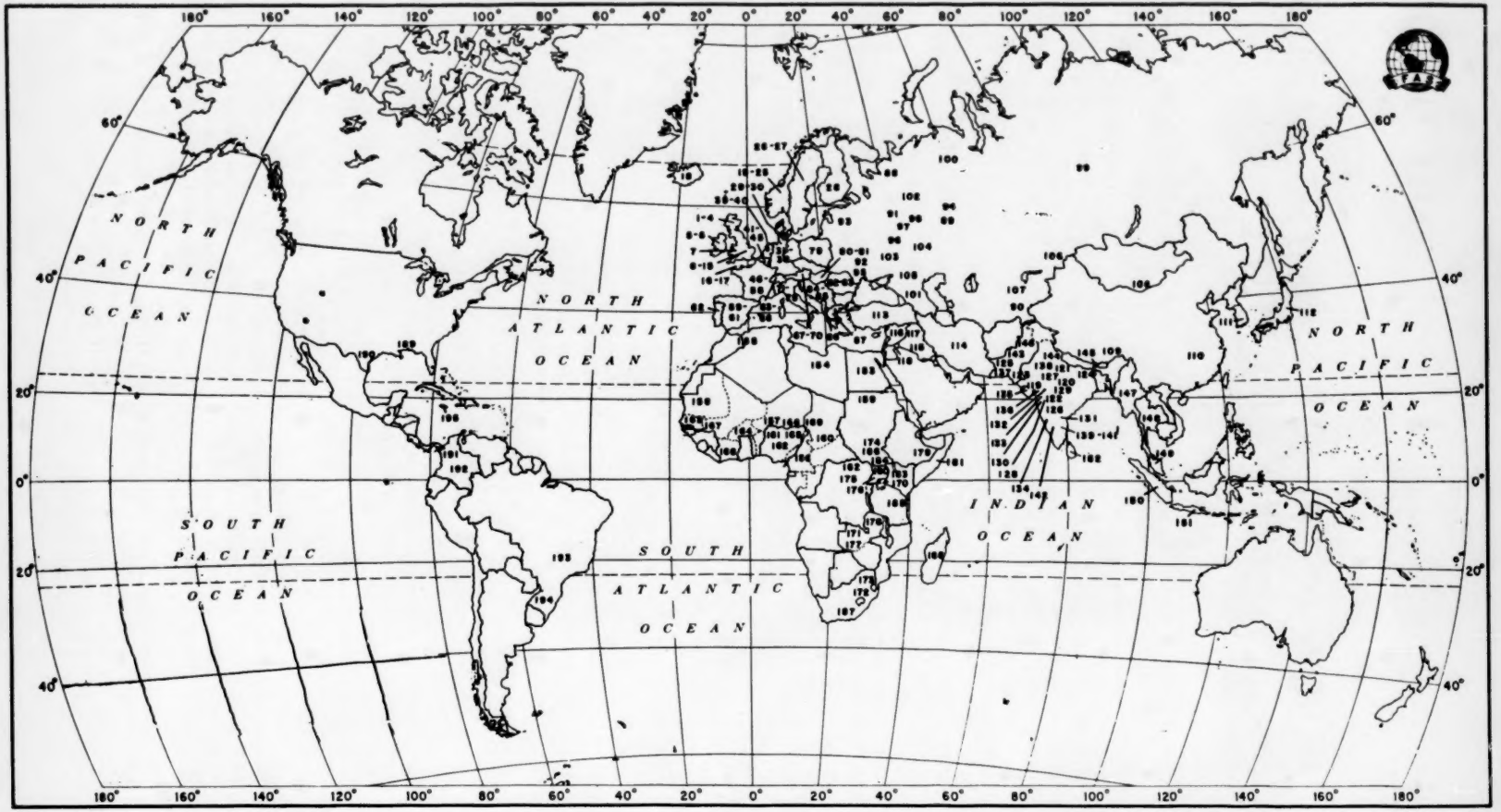

NORTH PACIFIC OCEAN
NORTH ATLANTIC OCEAN
NORTH PACIFIC OCEAN
SOUTH PACIFIC OCEAN
SOUTH ATLANTIC OCEAN
INDIAN OCEAN
180° 160° 140° 120° 100° 80° 60° 40° 20° 0° 20° 40° 60° 80° 100° 120° 140° 160° 180°
60°
40°
20°
0°
20°
40°

PLACES OF ORIGIN OF CATTLE BREEDS

Figure 8

Map showing the approximate places of origin of many of the breeds of cattle of the world. Some, while not fully recognized as breeds, are distinct local types. In other cases, more than one breed is covered by a number. The numbers shown on the map correspond to the numbers for the respective breeds set forth below.

Europe

Scotland: 1—Aberdeen-Angus; 2—Galloway; 3—Highland; 4—Ayrshire.

Ireland: 5—Dexter; 6—Kerry.

Wales: 7—Welsh Black.

England: 8—Hereford; 9—Beef Shorthorn; 10—Devon; 11—Sussex; 12—Dairy Shorthorn; 13—Lincoln Red Shorthorn; 14—Red Poll; 15—South Devon.

Channel Islands: 16—Guernsey; 17—Jersey.

Iceland: 18—Iceland.

Norway: 19—Red Polled Østlan; 20—Black-sided Trondheim and Nordland; 21—Telemark; 22—South and West Norwegian (including Lyngdal, Vestland Polled, Vestland Fjord); 23—Døle; 24—Red Trondheim; 25—Norwegian Red-and-White.

Sweden: 26—Swedish Red-and-White; 27—Swedish Polled.

Finland: 28—Finnish.

Denmark: 29—Red Danish; 30—Black-Pied Danish (including Black-Pied Jutland, Friesian).

Germany: 31—Yellow Hill (including Franconian, Glan-Donnersberg, Lahn, Limpurg); 32—German Brown (including Allgau, Murnau-Werdenfels); 33—German Red (including Bavarian Red, Harz, Odenwald, Sechsamter, Siegerland, Silesian Red, Waldeck, Vogelsberg, Vogtland); 34—Small Spotted Hill (including Hinterwald, Vorderwald); 35—Black-Pied Lowland (including East Friesian, East Prussian, Jeverland, Wesermarsch); 36—Red-Pied Lowland (including Lower Rhine, Red-Pied East Friesian, Breitenburg, Wilstermarsch, Red-Pied Westphalian); 37—Red East Friesian; 38—Angeln.

Netherlands: 39—Holstein-Friesian; 40—Groningen.

Belgium: 41—Blue; 42—Red West Flemish; 43—East Flemish Red Pied; 44—Campine; 45—Herve.

France: 46—Normandy; 47—Charollais; 48—Eastern Red Pied; 49—Limousin; 50—Maine-Anjou; 51—Parthenay; 52—Gascony; 53—Salers; 54—Aubrac; 55—Armorican; 56—Blond Pyrenian; 57—Breton Black-Pied; 58—Garonne.

Spain: 59—North Spanish (including Galician, Asturian, Leonese, Santander, Pyrenean); 60—Central and Southern Spanish (including Alvia, Extremadura, White Caceres, Marucho, Murcian, Serrana, Zamora, Andalusien); 61—Toro de Lidia.

Portugal: 62—Portuguese (including Alemtejo, Aroucesa, Bajocara, Borrosa, Jarmelo, Minho, Miranda, Ribatejo, Turino).

Switzerland: 63—Brown Swiss; 64—Simmental; 65—Herens; 66—Fribourg.

Austria: 67—Pinzgau; 68—Light Alpine (including Murboden, Blond, Waldviertel); 69—Brown Mountain; 70—Oberienntal.

Italy: 71—Chiana; 72—Romagnola; 73—Marche; 74—Maremma; 75—Piedmont; 76—Modena; 77—Grey Alpine; 78—Grey Adige.

Poland: 79—Polish Red.

Hungary: 80—Hungarian Red Spotted; 81—Hungarian Steppe.

Romania: 82—Romanian Steppe; 83—Romanian Mountain.

Yugoslavia: 84—Busa; 85—Yugoslav Steppe.

Albania: 86—Albanian.

Greece: 87—Greek Shorthorn.

U.S.S.R.

88—Kholmogor; 89—Tagil; 90—Aulie-Ata; 91—Yaroslavl; 92—Ukranian Red; 93—Latvian Red; 94—Suksun; 95—Ukranian Whitehead; 96—Gorbatov Red; 97—Yurino; 98—Istoben; 99—Siberian; 100—Pechora; 101—Caucasian; 102—Kostroma; 103—Tambov; 104—Bestuzhev; 105—Ukranian Grey; 106—Kalmyk; 107—Kirgiz.

Central and Eastern Asia

108—Mongolian; 109—Tibetan; 110—Chinese Yellow (including North China and South China types, with variations in each type); 111—Korean; 112—Japanese (three types of Wagyu[11]).

Near East

113—Anatolian (including Black and Red); 114—Persian; 115—Iraqi; 116—Oksh; 117—Kurdi; 118—Jaulan.

India and Pakistan

119—Kankrej; 120—Kenwariya; 121—Kherigarh; 122—Malvi; 123—Tharparkar; 124—Bachaur; 125—Bhagnari; 126—Gaolao; 127—Hariana; 128—Krishna Valley; 129—Mewati; 130—Nagori; 131—Ongole; 132—Rath; 133—Dangi; 134—Deoni; 135—Gir; 136—Nimari; 137—Red Sindhi; 138—Sahiwal; 139—Amrit Mahal; 140—Hallikar; 141—Kangayam; 142—Khillari; 143—Lohani; 144—Ponwar; 145—Siri; 146—Dhanni.

Southeast Asia and Pacific

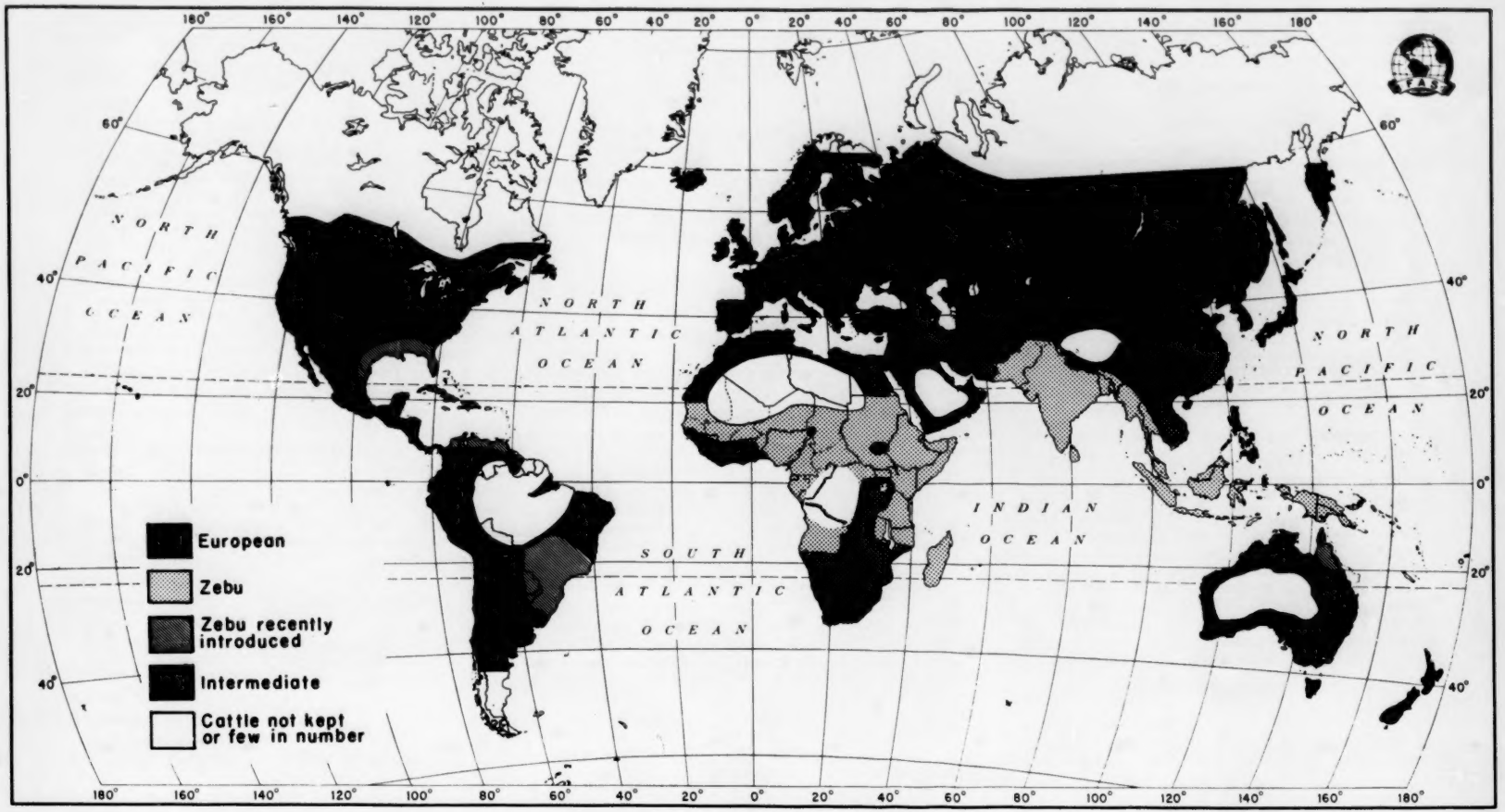
147—Burmese; 148—Thai; 149—Thai-Kedah; 150—Sumatran; 151—Indonesian; 152—Sinhala.

Africa

153—Egyptian (including Damietta, Baladi, Saidi, Maryuti); 154—Libyan; 155—Brown Atlas; 156—Adamawa; 157—Azaouak; 158—Maure; 159—Northern Sudan; 160—Shuwa; 161—Sokoto; 162—Nigerian Fulani; 163—Senegal Fulani; 164—Sudanese Fulani; 165—White Fulani; 166—M'Bororo; 167—N'Dama; 168—West African Shorthorn; 169—Kuri; 170—Ankole; 171—Barotse; 172—Basuto; 173—Nguni; 174—Nilotic; 175—Nioka; 176—Nganda; 177—Tonga; 178—Angoni; 179—Boran; 180—Bukedi; 181—Galla; 182—Lugware; 183—Nandi; 184—Southern Sudan Hill; 185—Tanganyika; 186—Toposa-Murle; 187—Africander; 188—Madagascar.

The Americas

189—Brahman; 190—Santa Gertrudis; 191—Romo Sinuano; 192—Blanco Orejinegro; 193—Indubrazil; 194—Caracu; 195—Jamaica Hope.



DISTRIBUTION OF THE TWO BASIC TYPES

Figure 9

Map showing the approximate distribution of the two major basic types of cattle, i.e., European-type humpless cattle (*Bos taurus*), and Zebu or humped cattle (*Bos indicus*), and of the intermediates between these two basic types.

last 50 to 75 years or so, for in some portions of the areas so marked, humpless European-type stock no doubt still prevail. Also, some intermediate types have no doubt developed in these areas.

Boundaries between types are only approximate since, in many areas, precise information is either not available or is not at hand to indicate exactly where dividing lines should be.

No attempt has been made in this map to show distinctions in types of production since two or more production types often co-exist within either of the basic types or the intermediates.

For these several reasons the map is limited to a portrayal of the distribution of the basic and the intermediate types. Granting some errors in the placement of the borderlines between types, it does give a good picture of the manner in which cattle have found their way around the world in the service of man.

Several factors have no doubt affected the way in which the present distribution came about.

It is rather easy to understand how the European-type humpless cattle spread over the land masses of Europe and Asia, and into northern Africa. It is also easy to imagine how Zebu cattle found their way into southeastern Asia and on to the islands of the South Pacific. Intermingling of the two types also took place, quite naturally, wherever they came in contact across Asia and the Near East. Both Zebu and intermediate types apparently found their way across the present land bridge between Asia and Africa and quite probably across the former land bridge at the southern end of the Red Sea. Many migrations of African tribes, further crossing between the two types, and the formation of new breeds in areas of geographic isolation have no doubt contributed to the rather curious distribution of basic types and intermediates in Africa.

European breeds were taken by European settlers to the new lands—North America, South America, Australia and New Zealand—and they still dominate the livestock scene almost entirely in most portions of these regions of the world. However, in recent times, Zebu cattle have come to occupy major places in the cattle industries in the Gulf Coast region of the United States, in portions of Latin America, and in northeastern Australia.

In general, we have come to regard the European breeds as being adapted to temperate climates, and the Zebu breeds as being adapted to the tropics and subtropics. Although the search for the basic reasons for the physiological differences in the reactions of Zebu and European-type cattle to heat and other environmental factors is only just beginning, it is clear that real differences do exist. On the other hand, there are substantial variations within each basic type in ability to withstand climatic conditions of different kinds. Also, among the many intermediate types and breeds that have evolved from interbreeding of European and Zebu types, there are no doubt many combinations of the characteristics contributed by the two basic types.

So the distributions of types in the tropical and subtropical portions of the world, as set out in Figure 9, reflect in large part natural migrations of men and cattle along overland routes, overseas migrations of men in which they took with them the kinds of cattle available in their native lands, and the presence of Zebus or types with substantial portions of Zebu blood in most tropical and subtropical areas, except those portions of the American tropics where descendants of early Spanish cattle still prevail. Thus, while the distribution pattern reflects the generally superior adaptability of Zebus to hot climates, other factors have contributed to the development of that pattern.

Earlier, reference was made to the complicated situation with regard to types and breeds of cattle in Africa. For purposes of the map in Figure 9, only three basic types—humped, humpless and intermediate—were recognized for Africa, as for other regions. However, the situation is more complex, as shown by the detailed analysis published recently by Mason and Maule[6]. These authors recognize two types of "intermediates" in East Africa—the Sanga,

with a small cervico-thoracic hump, and the Intermediate, in which are grouped those breeds and types intermediate to the Sanga and the "true" Zebu.

The Sangas appear to have been derived from crosses between Zebu and humpless cattle, for as in such crosses, the hump is reduced in size and advanced towards the neck. However, the Sanga types appear to be older than the East African Zebus, and Mason and Maule postulate that they may have emerged from an earlier wave of Asiatic Zebus, crossed with humpless cattle that appear to have been present, at least in northeast Africa, or that they may represent a stage in evolution of a humped from a humpless type.

Based on their observations, Mason and Maule[6] have published a very interesting and useful map of the distribution of cattle types in eastern and southern Africa. Owing to the different grouping, and the inclusion of more local types, that map presents a more detailed picture, and one which differs somewhat from the more generalized map shown in Figure 9. However, the broad picture is essentially the same.

If one could trace back from all present-day types of cattle to their ancestral roots in the original domestications, one would no doubt have a fantastically intricate story—intricate in respect of man's own migrations and the ways in which cattle were associated with him in those migrations—intricate in the way the multiplicity of breeds reflects many separate lines of development under widely differing environmental conditions and the intermingling of types and breeds emerging from these separate lines of development. Here is a story that may be as intricate as man's own history, and, in any event, closely related to that of man's recent development. The term "recent," is used advisedly because the period since cattle were domesticated, *i.e.*, the last 5,000 to 6,000 years, is short indeed compared with the 1,750,000 or more years since *Zinjanthropus* first emerged as a being with something more than animal intelligence, *i.e.*, the ability to fashion and use tools (Leakey[4]). But much of that story can never be written because many of the basic facts were never recorded.

Even now, our knowledge of the world's cattle is remarkably scant. Only in recent years have serious efforts been made to bring together comprehensive information regarding the cattle in some of the major regions of the world[2, 3, 6, 7, 9], or even to prepare lists of the breed names used in the various countries[5]. When one considers that the 889,500,000 cattle in the world constitute the most important single class of livestock serving man, it is surprising that we know so little about so many of those cattle.

Conclusion

Granting that much more information is needed before all the local types and breeds of cattle in the world can be classified with precision, or before the boundaries between the areas occupied by the major types can be defined with equal precision, this map showing the distribution of the humped, humpless and intermediate types of cattle is presented as one small step in the study of the ecology of cattle. It is hoped that it will be of value to those interested in cattle breeds and their distribution, in animal climatology, and in the improvement of cattle through breeding.

Also, since the map in Figure 9 must be regarded as a first effort rather than a finished product, it is hoped that it will provide a base upon which many others can build, filling in gaps so that we may have really adequate knowledge of the physical and physiological characteristics of cattle, and of the way they are distributed in all areas of the world where cattle are used.

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MULTIPLE SIRE MATING IN SWINE

III. Factors Influencing Multiple Paternity

LAVON J. SUMPTION AND JAMES C. ADAMS*

POSSIBLE values of multiple sire mating were proposed by Sumption *et al.*[10] Data showing substantial individual variation in mating behavior and in selective fertilization have been reported[5,9]. Considerable significance was attached to the possibility of increasing the variety of genetic recombinants where multiple paternity occurs in populations of limited size. If multiple sire mating is to warrant attention as a breeding method from the standpoint of increasing recombination potential, the following conditions must be satisfied within any population where it is applied:

1. Multiple mating must occur frequently.
2. The fertile interval during estrus must be sufficiently long to afford the opportunity for multiple paternity.
3. A high incidence of multiple paternity must ensue.

Evidence of the frequency of multiple paternity and the conditions under which it is likely to occur will be presented in this paper.

Experimental Procedure

Experiment Ia—random mating

Ten Duroc sows were exposed daily and simultaneously to one sire of each of five breeds (Duroc, Hampshire, Minnesota No. 1, Yorkshire and Line X, a local strain) until all the sows were mated. Boars used for this and all subsequent experiments were fertile, as determined by immediately previous natural mating. The frequency of successful mating was recorded for each sire and dam during two-hour mating periods twice daily, eight hours apart. Time of mating in relation to onset of estrus was not standardized, but notes were kept on the actual time when mating occurred. Mating success of boars was determined by intromission and ejaculation (ejaculation is invariably associated with contraction of the anal musculature). The paternity of progeny was determined by color markers and differences in body form, as previously described[9]. Therefore, in the cases of multiple paternity some limited information could be gathered on the length of the fertile period during estrus.

Experiment Ib—random mating

Sixteen gilts of three breeds were exposed simultaneously to 12 sires (four each of the Duroc, Hampshire and Yorkshire breeds) during a daily two-hour observation period until all gilts were mated. The number of matings per sire that resulted in vaginal ejaculation was recorded.

Experiment II—controlled multiple mating

Each of twelve Duroc gilts was mated in rapid succession and rotated order to one boar of each of the Berkshire, Duroc, Hampshire, Minnesota No. 1 and Yorkshire breeds. The maximum interval between the first and last mating was 65 minutes ($\bar{x}$ = 39 minutes). The extent of multiple paternity was determined by the color markings of the progeny. Effects of mating order could be examined because of the plan of rotated mating which was followed.

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Experiment III—double mating

Twenty Duroc gilts were mated during the first day of estrus to a Duroc sire and a sire of a second breed; on the second day of estrus each dam was remated to the Duroc sire and also to a sire of a third breed. The primary purpose was to produce purebred and crossbred pigs in the same litter. Incidental to this major objective, it was possible to investigate whether matings that occurred as much as 24 hours apart were effective.

Experiment IV—single mating

Ten Duroc gilts were mated on the first day of estrus to a sire of one breed and on the second day to a sire of another breed. The sole purpose was to determine whether two matings that occurred 24 hours apart were effective. All matings for Experiments II, III and IV were made during a period of six consecutive hours each day.

Results

Multiple mating occurs frequently when boars are allowed free access to a group of females (Table I). Twenty-one of the 26 gilts observed were mated two or more times during the periods of exposure. The average mating frequency per female was 3.54. Subsequent matings were made by different sires, unless copulation was disturbed early in ejaculation. In the latter case, there was a strong drive for a boar to remate the same female immediately and attain some minimum time of ejaculation that is apparently related to sexual satiation of the boar.

The frequency of multiple paternity was high. Taking all four experiments from which data could be drawn, 36 of 52 litters (61 percent) were sired by more than one boar (Table II). Multiple paternity was more frequent when matings occurred in a shorter time interval. When the data from Experiment IV are excluded, 81 percent of the litters were multiple sired.

Some general ideas about the period during estrus in which fertile matings by different sires can occur were gained from Experiments Ia, III and IV. The fertile mating period was somewhat shorter than the period during which females were actually receptive in Experiment Ia (Table III). Exceptions were female 122, that accepted only one mating during a two-day estrus, and female 219, for which the effective and the total periods were the same. The effective periods involved under the existing experimental conditions did allow for the occurrence of multiple paternity in six of the 10 litters observed. One litter in Experiment IV was sired by two boars (Table II), indicating that matings 24 hours apart were actually effective. Effective matings occurred 25 hours apart in two of the 20 litters in Experiment III (Table II).

From a physiological and an operational standpoint, it is important to know if the order of mating has any influence on the frequency of representation of a given male in a multiple sired litter (Tables IV and V). Experiment Ia did not provide clear results because of variations in the frequency of mating, but no striking effects of mating order were evident (Table IV). There is a considerable range in the number of progeny per mating in individual litters shown in Table V. The data are arranged by order of mating without regard to specific sires. No obvious trend was exhibited in this case where all matings were restricted to a relatively short period of time.

Discussion

Evidence was advanced previously to show that both mating frequency and fertilization rate were sufficiently non-random under multi-

TABLE I. Frequency distribution of matings per female under random mating during one estrus period

	No. of matings						
	1	2	3	4	5	6	7
	No. of females						
Experiment Ia	1	1	1	3	1	2	1
Experiment Ib	4	4	2	2	2	0	2

ple sire mating to lead to a significant degree of differential reproduction[9]. The possibility of selecting for mating efficiency by using multiple sire mating was proposed. The magnitude of multiple mating is emphasized further in this paper. More recent studies have yielded extremes in female mating frequency that exceed those in Table I by more than twofold[5]. As a result of multiple mating, multiple paternity was a common occurrence under both controlled and random breeding. It should be clear that with the exception of inducing competitive mating among males, possible preferential mating by both sexes and selective fertilization, the useful upper limit of multiple mating is determined by the number of living progeny produced in each litter.

The fertile period during estrus is sufficiently long so that multiple paternity can occur readily. No attempt was made to establish the absolute limits of this period because first exposure of females was not standardized in relation to the onset of estrus. It is likely that differences in the fertile period may be related to variations in events during estrus. First of all, the length of estrus is quite variable[3]. Furthermore, the time of ovulation during estrus, in relation to the onset of receptivity, exhibits marked genetic differences. For example, Large White sows ovulated from 18 to 36 hours after the onset of estrus, as contrasted with the interval of 42 to 54 hours for the Large Black[3]. Ovulation occurred during approximately 6.5 hours of the estrus period.

The available evidence indicates that the relative number of progeny per sire was not greatly influenced by the order of mating (Table V). This result agrees with findings in the double mating experiments of Lush *et al.*[7] There is no evidence of the phenomenon that is so common in poultry[2] where the latest ejaculate is usually responsible for fertilization, probably because of the short period of ovulation in swine. Sperm placed in the tract immediately after the onset of estrus can fertilize ova that are shed some 48 to 54 hours later. However, Burger[3] suggests that the optimum time for mating is in the first 12 to 24 hours of estrus. Matings during and after the estimated time of ovulation were progressively less successful due to the known reduction in viability of ova[3] coupled with the possible problem of "capacitation" of sperm[3]. A further reason for the lack of a consistent effect of mating order in these studies may be that breeding seldom occurred at the extremes of receptivity. Combining these results with an earlier report[9], it is suggested that selective fertilization as a physiological factor is the primary cause of variations in paternity rather than order of mating as a factor in the timing of sperm delivery. This proposal depreciates the importance of extremes in social dominance and sex drive of sires, except as these traits are associated with selective fertilization. It is possible that a male that mates a female somewhat late in estrus, because of his low social order or sex drive, could sire a majority of her progeny if he should possess physiologically dominant sperm.

TABLE II. Distribution of single and multiple sired litters in studies where females were exposed to boars of more than one breed

Mating method	No. of effective sires				
	1	2	3	4	5
	No. of litters				
Single mating, Expt. IV	9	1	—	—	—
Double mating, Expt. III	2	16	2	—	—
Random mating, Expt. Ia	4	3	3	0	—
Controlled multiple mating, Expt. II	1	2	7	1	1

TABLE III. Observed length of the fertile mating period under random mating (Experiment Ia)

Female no.	Observed total mating period	Fertile mating period	Total no. of matings/female	Total no. of sires mating each female	No. of effective sires
	hours	hours			
110	28.7	11.3	6	4	2
122	—	—	1	1	1
124	27.7	—	3	2	1
132	10.3	0.5	5	4	3
133	39.4	1.1	7	4	3
217	24.6	9.2	4	3	3
219	11.9	11.9	2	2	2
419	49.0	—	6	4	1
508	36.7	8.2	4	3	2
527	18.9	—	4	3	1

TABLE IV. Distribution of progeny under random mating (Experiment Ia)

Litters	Sequence of matings (first through fourth)			
	1	2	3	4
	No. of progeny			
110	1	5	0	0
122	8	—*	—	—
124	7	0	—	—
132	2	2	1	0
133	3	4	2	0
217	2	2	2	—
219	4	5	—	—
419	0	6	0	0
508	0	6	1	—
527	0	9	0	—

* No mating.

Normal mating behavior patterns of both males and females augment the frequency of multiple paternity. Observations of group behavior reveal that ordinarily a number of boars will mate with each female shortly after the onset of estrus[5]. Frequent mating continues throughout estrus if a female is placed with either one[3] or several[9] sires. When females are exposed simultaneously to a group of boars, each boar tends to seek out and mate a "new" female rather than immediately re-mating the same individual. This finding agrees with the extensive studies of bulls in which the frequency of ejaculation was increased by the frequent presentation of new stimulus animals[6]. Thus, under multiple sire mating the sexual behavior of boars generally will assure the presence of viable sperm from two or more sires at the site of fertilization.

All of the observations in this work emphasize the ease with which multiple sired litters can be obtained. As compared to conventional single sire mating, genetically distinct full-sib families are increased in number but proportionately reduced in size by multiple paternity. An array of small maternal half-sib families are created in the process. The *average* size of paternal half-sib families may not change materially. That is, boars will tend to sire a small sample of progeny in a larger number of litters. There may be, however, *more variation* in the number of progeny per sire because of the behavioral and physiological influences already discussed. Thus, the genotype of a female may be sampled simultaneously in combination with more than one sire. Furthermore, it is possible to sample the genotypes of a group of sires in combination with more females than can be accomplished in the same size population with single sire mating.

A basic question for future study is whether the greater diversity of sire-dam combinations that can be generated by multiple sire mating might have a significant influence on the rate of genetic improvement[10]. The increased diversity of combinations is obtained only at the expense of the number of progeny from any single combination. Decisions on a breeding system, with respect to recombination potential, must reckon with the optimum relationship between sample size and the number of samples of different parental genotypes that can be obtained in a population of constant size. The approach should be influenced by the magnitude of genetic differences among both sires and dams. The significance of designing breeding systems that are maximally efficient in releasing genetic variability through recombination is clear from the work of Anderson[1], Dobzhansky *et al.*[4], and Suneson[11].

Summary

Results of five experiments involving 29 sires and 58 females were examined to evaluate the frequency of multiple paternity under conditions of multiple sire mating. Multiple mating of the majority of females is common. The fertile mating period during estrus is sufficiently long so that multiple paternity occurred in 69 percent of all litters. Order of mating as a factor in timing of sperm delivery apparently has less effect on the relative number of progeny per mating per sire than the physiological phenomenon of selective fertilization. The normal mating behavior pattern of boars augments multiple paternity because a different female provides a greater sexual stimulus for subsequent matings than the female previously mated. The role of multiple sire mating in ex-

TABLE V. Distribution of progeny as affected by order of mating (Experiment II)

Litters	Sequence of matings					Interval between first and last mating
	1	2	3	4	5	
	No. of progeny					Minutes
10	2	6	3	0	—*	55
20	4	0	7	2	0	25
30	0	8	0	1	1	35
40	1	1	2	9	1	30
50	7	1	0	0	2	19
60	0	0	0	0	8	40
70	0	0	10	0	1	65
80	0	0	9	0	3	35
90	0	1	0	6	1	55
100	1	2	1	0	9	45
110	8	0	0	2	1	35
120	3	0	3	1	0	30
Total progeny	26	19	35	21	27	
Av. no. progeny/mating†	2.17	1.58	2.92	1.75	2.45	

* No mating.
† P = >.10<.20.

tending recombination potential in swine breeding is discussed.

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WE REGRET THE DELAYS

Every effort is being made to put the *Journal* back on a current publication basis. However, it will be several months before this can be fully achieved.

During the past year we have been beset with difficulties of many kinds—financial and otherwise—which are being slowly but surely rectified.

To members, subscribers, and authors who have submitted manuscripts for publication we ask your patience for a little while longer and wish to express our appreciation for your endurance. A great deal of progress has been made in the business of catching-up and there is no reason at present to suppose that it will not continue. But we still have quite a long stretch ahead.

—B.C.L.

TIMIDITY AND AGGRESSIVE BEHAVIOR OF THE RAT

A Demonstration of the Functions of Genetic Strain

JOSEPH ROSEN*

IN recent years there has been a steadily growing interest in that area of psychological research known as psychogenetics or behavior genetics. Comprehensive reviews of the literature are to be found in Fuller[1] and Hall[2].

Although there have been a number of studies concerned with differences in aggressiveness among various strains of mice, the purpose of the present investigation was to determine whether there are any inherited differences in both timidity and competitive aggression in two strains of domesticated laboratory rats. Competitive aggression refers to aggressive behavior elicited by the presence of a goal presumed to be attractive to several subjects at the same time.

Method

Subjects

Ten male Wistar albino rats and 14 male non-agouti black hooded rats were used in this study. They were housed in individual cages and maintained on an *ad libitum* diet of food and water until 75 days of age.

Apparatus

The Aggression Test Box was a 48 × 15 × 12 inch black wooden box with a wire-mesh lid. A starting cage was placed at the right end of the box. This cage had a guillotine door which could be raised vertically through an opening in the wire-mesh lid. A metal feeding cylinder, $2\frac{1}{4}$ inches high and $1\frac{1}{4}$ inches in diameter, was fastened in the center of the box.

Procedure

The experiment was divided into three distinct phases:

Drive Cycle: When the animals were 75 days of age, all food was removed from their cages. From 76 to 89 days of age, they were given a single daily one-hour feeding of wet mash. Water was always freely available.

Familiarization: This period provided a transition to the aggression test period during which the animals were required to fight for control of and to eat from the feeding cylinder in the Aggression Test Box. It also allowed the rats to explore and become familiar with the inside of the apparatus. Each rat was given a single three-minute trial for five consecutive days beginning at 90 days of age.

Five seconds after being placed inside the starting cage of the box, the guillotine door was raised and subjects were permitted to enter the rest of the apparatus to eat from the cylinder. The animals were provided with their regular one-hour feeding at the completion of all daily trials.

Aggression Test: The 14 hooded rats were randomly divided into seven pairs of competitors and the 10 albino rats into five pairs of competitors. The same pair of rats fought against each other every day of the test period. Each pair was given seven trials on consecutive days starting at 95 days of age. Each trial lasted either until the occurrence of the first vigorous fight or for five minutes without fighting. The fighting response latencies were used as the measure of aggressive behavior[4]. If the

*This investigation was performed at the Department of Psychology, University of Toronto, Toronto, Canada. The author is now with the Division of Behavior Studies, Roscoe B. Jackson Memorial Laboratory, Bar Harbor, Maine.

pair of animals did not fight during a trial, they were assigned a latency of 300 seconds.

Five seconds after each pair of competitors was placed inside the starting cage of the apparatus and the wire-mesh lid lowered, the guillotine door was raised to permit the rats to race toward the feeding cylinder. Animals were given their regular feeding of mash at the end of all daily competitions. Water was *ad libitum*.

Results

The timidity score of each animal was based on the mean of the emergence-from-cage response latencies during the five-day familiarization period. The mean latency score for the black hooded rats was 13.62 seconds, and for the albinos 18.54 seconds. The *t*-value of .871 was not significant.

There was also no significant difference in the mean fighting response latencies between the two strains as tested by the Mann-Whitney U Test (U of 15).

Discussion

Although this investigation has disclosed no genetic differences in timidity and competitive aggression between the non-agouti black hooded strain and the Wistar albino strain of rats, the present findings may be of importance from other aspects. First, the findings relating to timidity confirm those reported earlier by Keeler[3]. He reported a mean of 54 seconds for albino rats and a mean of 48 seconds for the black hooded rats on a refined emergence-from-cage test. Secondly, recently completed research by the author (unpublished data) has shown that the black hooded rats are significantly more dominant in competitive feeding situations than the albino rats. These differences in dominance can be attributed solely to differences in genetic background since both strains were given the same treatment throughout the course of the experiment. Since no genetic differences in competitive aggression were found, it would appear that aggressiveness and dominance represent two distinct patterns of social behavior. Such a position is in accord with the findings of Warren and Maroney[5] in that the relationship between social dominance and aggression may be species-specific.

This experiment has also shown that even under conditions of competition, aggressive behavior in the form of vigorous fighting is difficult to elicit in these two strains. With the hooded rats, vigorous fighting occurred in 42 percent of the trials, while with the albinos it occurred in only 31 percent of the trials. Although vigorous fighting occurred infrequently, the animals attempted to control the goal object by active pushing and shoving, butting, and "rooting" behavior.

Summary

This experiment has demonstrated no genetic differences in timidity and competitive aggression between the non-agouti black hooded and Wistar albino strains of rats. Although there were no significant differences in aggression between these two strains, previous research has disclosed significant genetic differences in terms of social dominance. Social dominance and aggressiveness may therefore represent two distinct patterns of social responsiveness.

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INHERITANCE OF SEED-COAT MOTTLING IN LOTUS TENUIS

ROBERT R. SEANEY*

NARROW-LEAF birdsfoot trefoil, *Lotus tenuis* Waldst. and Kit. ex Willd., is a perennial legume used as a pasture and hay crop principally in New York, California and Oregon. Within plant populations from these states, plants with either mottled or non-mottled seed can be found. This mottling (Figure 10) appears as small black spots of varying size on a brown background. Mottling of seed from a single plant can vary from a few small dots on some seeds to larger black patches on others. The inheritance pattern of this character makes it useful as a genetic marker for identification of varieties and populations in breeding materials.

Levy[2] described seed mottling in the closely related species *L. corniculatus* L. ($2n = 24$) and noted mottling was absent on some seeds. Donovan[1] selected plants of *L. corniculatus* with and without the mottling character and in inheritance studies concluded that mottling was determined by disomic inheritance of two genes, with mottling dominant to non-mottling. He observed that environmental factors in the field varied the expression of mottling.

Materials and Methods

Plants used in this study were selected from an accession of *L. tenuis* received from California. Chromosome counts of all selected plants were $2n = 12$.

In all crosses, flowers were emasculated approximately two days before hand transfer of pollen from male to female plant. Plants were self-pollinated by rolling flower clusters between the fingers.

F_1 progenies were grown in both greenhouse and field, and F_2 and backcross progenies were grown outside in 3-inch pots placed on gravel.

Results

For study of segregation of mottling in S_1 progenies, 25 plants of *L. tenuis* were self-pollinated in order to find plants with some degree of self-fertility. As shown in Table I, six plants were found with moderate self-fertility. Self-fertility was expressed in terms of number of seeds set divided by number of flowers pollinated. Self-progenies of these six plants were grown and S_1 plants observed for seed mottling. Segregation ratios in S_1 progenies are shown in Table I.

Two plants were selected for crossing, plant A which had all S_1 progeny with mottled seed

TABLE II. S_1 and F_1 progenies of parent plants A and D

Plant	Classification of progeny	
	Mottled	Non-mottled
A selfed	103	0
D selfed	0	120
A × D	171	0

TABLE I. Self-fertility and S_1 segregation ratios for seed-coat mottling of six plants of *L. tenuis*

Plant	Classification of seed of S_1 plants		Self-fertility		
	Mottled	Non-mottled	Flowers pollinated	Seed set	Seed per flower
A	45		46	56	1.2
B	16		112	148	1.3
C	10		132	33	.25
D		30	77	152	2.0
E	34	12	275	301	1.1
F	10	2	160	77	.48

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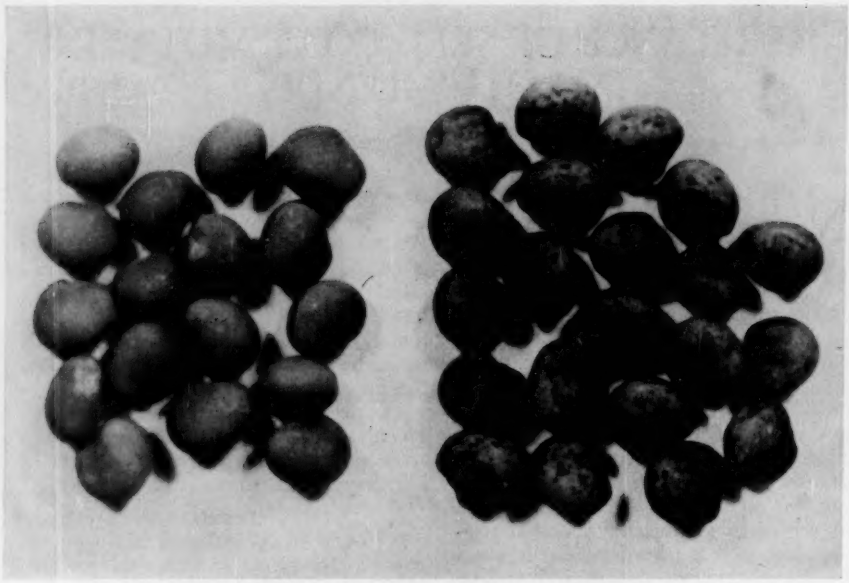

NON-MOTTLED AND MOTTLED SEED COAT

Figure 10

Seed of *Lotus tenuis* showing non-mottled (left) and mottled seed coat.

and plant D which had all S_1 progeny with non-mottled seed. These two plants were self-pollinated and intercrossed. All F_1 plants of the cross A × D had mottled seed (Table II).

Selfed and backcross progenies of nine F_1 plants from the cross A × D are shown in Table III. Applications of chi-square tests of the observed class frequencies in each self and backcross progeny indicate that F_2 progenies segregated in a ratio of three plants with mottled seed to one plant with non-mottled seed and that backcross populations segregated one with mottled seed to one with non-mottled seed.

These data indicate single-factor inheritance of seed-coat mottling, with mottled seed dominant to non-mottled.

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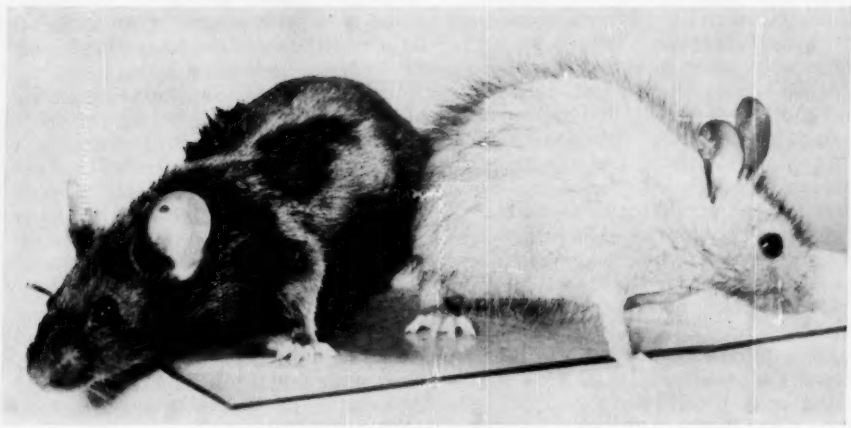
TABLE III. Self and backcross progenies of F_1 plants from the cross plant A × plant D

F_1 plant identification	Self-progenies of F_1 plants		Backcross of F_1 plants to plant D		Backcross of F_1 plants to plant A	
	Mottled	Non-mottled	Mottled	Non-mottled	Mottled	Non-mottled
AD–1	38	15	43	37	34	0
AD–2	28	6	15	18	30	0
AD–3	27	9	14	12	25	0
AD–4	61	19	27	24	19	0
AD–5	33	13	20	14	12	0
AD–6	26	10	18	16	21	0
AD–7	23	7				
AD–8	27	9				
AD–9	36	7				

OPOSSUM, A SEMI-DOMINANT LETHAL MUTATION

Affecting Hair and Other Characteristics of Mice

EARL L. GREEN AND STANLEY J. MANN*



NORMAL AND OPOSSUM MICE

Figure 11

Opossum mice have deficient coats of fur, very few vibrissae, and grow more slowly than normal mice. (C57BL/6J ♀ 850-2; Opossum ♂ 622-6.)

IN SEPTEMBER of 1957, a male mouse with very sparse fur was found in a litter of B6D2 F_1 hybrids (C57BL/6J ♀ × DBA/2J ♂) at the Jackson Memorial Laboratory. The aberrant type was dubbed "Opossum." The name has continued in use since it is the most apt name so far suggested. Breeding tests have shown that "Opossum" is due to a new dominant autosomal mutation, probably lethal when homozygous. It is in linkage group 5 and may be an allele of *Ra* (Ragged) or closely linked to it.

Opossum mice are deficient in fur and vibrissae, are smaller in size from birth up to three months of age and probably beyond, and have decidedly lower prenatal and postnatal survival rates than non-Opossum littermates. The symbol *Op*, Opossum, is suggested.

Description of Opossum

The following description applies only to Opossum heterozygotes, since homozygotes have not been found.

The adult Opossum heterozygote has a sparse coat of hair, fewer vibrissae,

*Roscoe B. Jackson Memorial Laboratory, Bar Harbor, Maine, and Department of Biology, Brown University, Providence, R. I., respectively. This investigation was supported in part by research grant E-162 from the American Cancer Society to the Jackson Memorial Laboratory, and in part by PHS research grant C-592 from the National Cancer Institute and by PHS training grant 2G-329 from the Division of General Medical Sciences, Public Health Service, to Brown University. The authors are indebted to Mr. Leslie Rinaldo for first discovering the "Opossum" mouse in a litter of B6D2 F_1 hybrids, to Mrs. Florence Leland for diligent assistance in carrying out the breeding tests, to Mrs. Priscilla Lane for pointing out how to distinguish agouti Opossum from non-agouti Opossum mice, and to Dr. Margaret C. Green for providing the linkage data in Table IV.

and smaller body size than non-Opossum mice. Whereas the normal pelage of the mouse consists of overhairs (monotrichs, awls, auchenes) and undercoat (zigzags)[2], the Opossum pelage lacks auchenes, lacks zigzags except for an occasional small patch on the shoulders, has some awls, and possibly has a full complement of monotrichs. However, the hairs are difficult to classify because they grow asynchronously after the first coat. The "special" hairs on the feet, tail, ears, and cheeks are present and appear normal. The posterior vibrissae on the upper lip are normal, but the anterior vibrissae and their follicles are completely missing. The sparse coat and the apparently narrowed snout, probably due to fewer vibrissae and follicles, suggested the name Opossum.

By 12 days after birth, Opossum mice are distinctly smaller and at weaning age they are about two-thirds the size of their normal sibs. The few which have survived and been used for breeding appear healthy and may live more than a year.

At birth, the Opossum heterozygotes may be recognized by the deficiency of vibrissae. A large portion of Opossum mice are dead or dying when found after birth. These are cyanotic and edematous. The edematous skin of dead Opossum mice has a stretched, glossy, membranous appearance, with red patches, probably due to superficial hemorrhages. The dying mice are usually edematous in the neck and shoulder regions. Both the dead and dying mice have abnormal amounts of fluid in the abdominal and pleural cavities.

The onset of pigmentation in the skin of Opossum is delayed because of a lag in growth of pelage hairs. By three days after birth, when the skin of normal mice turns dark and hairs begin to erupt through the surface, the skin of Opossum mice is still pink due to the sparse coat.

Breeding Tests

In matings to 11 females of several different inbred strains, the original Opossum male sired 135 mice, classified at weaning as 96 non-Opossum and 39 Opossum. This established that Opossum is inherited, that probably a single locus had mutated, that, if so, the original male was heterozygous, and that *Op* is dominant. The recovery of

TABLE I. Results of matings of Opossum and normal mice. Progeny classified at weaning

Mating		No. of matings	No. of litters	Normal		Opossum		Total
Dam	Sire			♀♀	♂♂	♀♀	♂♂	
1. Normal	× Opossum	11	26	46	50	16	23	135
2. Normal	× Opossum	8	27	39	49	2	4	94
3. Normal	× Opossum	7	20	24	27	4	8	63
4. Normal	× Opossum	6	24	46	37	4	7	94
5. Normal	× Opossum	18	43	62	86	11	16	175
Total		50	140	217	249	37	58	561
6. Normal	× Normal	3	13	44	25	0	0	69
7. Opossum	× Opossum	16	32	1	9	0	1*	11

Explanation of matings:

1. Original Opossum ♂ mated with ♀♀ from a variety of strains: C57BL/6J, DBA/2J, C58/J, C57BL/6-a^t (ELG data).
2. Matings of Opossum ♂♂ from mating 1 with C57BL/6J ♀♀ (ELG data).
3. Matings of Opossum ♂♂ from mating 2 with C57BL/6J ♀♀ (ELG data).
4. Matings of Opossum ♂♂ from mating 3 with C57BL/6J ♀♀ (ELG data).
5. Matings of Opossum ♂♂ from mating 3 and later generations with sibs and with C57BL/Ch ♀♀ (SJM data).
6. Matings of normal ♂♂ and ♀♀ from mating 1 (ELG data).
7. Matings of Opossum ♂♂ and ♀♀ from all generations (ELG and SJM data). The male marked *, No. 752–1, was classed as an Opossum shortly after birth, but by weaning age had a coat of fur very different from Opossum. In fact, the coat looked normal, but seemed to lack monotrichs. In test matings, he sired 40 normal and 0 Opossum progeny. He died unexpectedly before hair samples were examined.

fewer than 50 percent Opossum mice, as expected from matings of $+Op \times ++$, may be explained as a lowered viability of the Opossum phenotype, rather than as an abnormal segregation, or as two or more simultaneous mutations, or as incomplete penetrance of the Opossum gene.

Subsequent matings have confirmed the idea that Opposum is due to a dominant mutation. The data summarized in Table I show that matings of Opossum × normal mice produce both Opossum and normal (matings 1–5), that extracted normal × normal produce only normal, thereby eliminating the possibility of incomplete penetrance of the Opossum gene (mating 6), and that Opossum × Opossum matings are capable of producing normal progeny (mating 7). The Opossum females are such poor breeders that in 16 matings with Opossum males only 32 litters have been produced, and only 11 progeny have been weaned. With one dubious exception, all progeny of Opossum × Opossum matings have been normal. On the strength of these breeding results, Opossum is regarded as due to a dominant autosomal mutation which is probably lethal when homozygous. The symbol *Op* is proposed.

Viability of Opossum Mice

If Opossum is due to a completely penetrant, dominant, single-locus mutation, Opossum heterozygotes are clearly less viable than their non-Opossum siblings. Altogether 252 litters were produced from matings of normal females by Opossum males (Table II), in which 50 percent of the progeny should have been Opossum if the viability of Opossum were not depressed. Of 1,035 mice classified at weaning age, 150 were Opossum, and 885 were normal. The ratio of the number of Opossum to the number of normal mice, an estimate of relative viability, was 16.9 percent.

The mortality of Opossum mice is not wholly postnatal. Of the 1,001 mice born in matings 1–4 and 8–10, 955 lived long enough to be classified within a few days after birth. Two hundred and four were Opossum and 751 were normal (sexes combined), making a viability ratio shortly after birth of 27.2 percent. One of the authors (S.M.) made a special attempt to observe and classify the progeny of normal × Opossum matings within 10 hours after birth, to see if about one-half of the newborn were Opossum. In 43 litters, 268 mice were found within 10 hours after birth. Of these, 64 were dead or dying Opossum, 41 were viable Opossum, and 163 were normal (Table II, mating 5). The deficiency of Opossum mice is significant even at birth ($\chi^2 = 11.06$ exceeds $\chi^2(0.05, 1) = 3.84$). The viability ratio was 25.1 percent.

There appears also to be a deficiency

TABLE II. Viability of Opossum mice. Progeny classified at birth. Unc. = unclassified

Mating			At birth				At weaning			
Dam	Sire	No. of litters	Normal	Living Opossum	Dead or dying Opossum	Unc.	Normal	Opossum	Dead	Total
1–4 Normal	× Opossum	97	332	89	—	27	318	68	62	448
8–10 Normal	× Opossum	112	419	115	—	19	419	55	79	553
5 Normal	× Opossum	43 ♀♀	70	20	23	—	62	11	93	
		♂♂	93	21	27	—	86	16		
		Unsexed	—	—	14	—	—	—	—	
		Total	163	41	64	—	148	27	93	268
All matings		252	914	245	64	46	885	150	234	1269

of females among the Opossum mice at weaning. In matings 1–5, Table I, the ratio is 37 ♀ ♀ : 58 ♂ ♂ ; in matings 8–10, Table III, the ratio is 19 ♀ ♀ : 36 ♂ ♂. In both sets of data, the deviation from equal numbers is significant. However, at birth, the ratio in mating 5, Table II, was 43 ♀ ♀ : 48 ♂ ♂, suggesting that the depletion of the Opossum females is greater between birth and weaning. In fact, of 20 females viable at birth, 11 survived to weaning, whereas of 21 viable males, 16 survived to weaning.

Linkage of *Op* and *a*

Opossum mice resemble Ragged (*Ra*) mice, although Opossum heterozygotes have less fur than Ragged heterozygotes and more fur than the viable Ragged homozygotes. *Ra* is linked with *a* (non-agouti) in linkage group 5[1]. The linkage of *Op* and *a* was tested and it was found (Table III, matings 9 and 10) that *Op* and *a* are linked with 26.8 ± 2.1 percent of recombination in males in coupling and repulsion combined.

The classification of agouti (+*a*) and non-agouti (*aa*) among Opossum (*Op*+) mice was aided by viewing the cheek hairs immediately beneath the eyes with a hand lens. These hairs are clearly banded in agouti and not so in non-agouti mice. Not all Opossum mice could be classified as agouti or non-agouti by viewing the hair with a hand lens because the hair was often (17 cases in 47) too sparse. Eleven classified Opossum mice were tested by mating them with *aa* mice to see if they were agouti (+*a*) or non-agouti (*aa*). In every case the breeding results agreed exactly with the classification by viewing the cheek hairs.

Since *Op* resembles *Ra* and is linked with *a* with about the same probability of recombination as *Ra*, it would be desirable to test for the linkage or allelism of *Op* and *Ra*. This test is not immediately practical, as discussed in the next section. The question can be approached indirectly by determining if *Op* and *Ra* have similar linkage relationships with other markers in link-

TABLE III. Test for linkage of the *a* locus and the *Op* locus. Progeny classified at weaning. c = recombination percentage, s_c = standard error of c

Mating Dam	Mating Sire	No. of matings	No. of litters		++	+*a*	*Op*+	*Opa*	*Op*?	Total	c ± s_c
8. ++	*Opa*	4	5	♀ ♀	9	—	1	—	—		
++	+*a*			♂ ♂	5	—	5	—	—		
				Total	14	—	6	—	—	20	
9. +*a*	*Opa*	18	76	♀ ♀	108	41	1	9	8		
+*a*	++			♂ ♂	99	41	8	12	9		
(repulsion double backcross)				Total	207	82	9	21	17	336	28.5 ± 2.5
10. +*a*	*Op*+	9	31	♀ ♀	16	39	—	—	—		
+*a*	+*a*			♂ ♂	10	51	2	—	—		
(coupling double backcross)				Total	26	90	2	—	—	118	22.0 ± 4.0

Explanation of matings:
8. Opossum ♂ mated with four normal ♀ ♀ of C3HeB/FeJ (ELG data).
9. F_1 Opossum ♂ ♂ from 8 mated with C57BL/6J ♀ ♀ (ELG data).
10. Backcross Opossum ♂ from 9 mated with C57BL/6J ♀ ♀ (ELG data).

TABLE IV. Three-point test with *a*, *pa*, and *Op*. Progeny classified at weaning. (Data of Dr. Margaret C. Green)

Mating Dam	Mating Sire	Progeny *Op*++	+*apa*	*Op*+*pa*	+*a*+	*Opapa*	+++	*Opa*+	++*pa*	Total
11. +*apa*	*Op*++	1	22	0	5	1	13	0	0	42
+*apa*	+*apa*									

age group 5. It is already known [1,3] that *Ra* is on the opposite side of *a* from *pa* (pallid). The results of a backcross of triple heterozygotes to non-agouti pallid mice are shown in Table IV, mating 11. The absence of the *Opa*+ and ++*pa* classes shows that these are probably the double recombination classes, and the order is therefore *Op-a-pa*. *Op* and *Ra* are thus on the same side of *a*.

Allelism of *Op* and *Ra*

A test of allelism of *Op* and *Ra* requires recovery of the compound Opossum-Ragged mouse. Attempts to do so have so far been unsuccessful. It is unlikely that the compound *Op-Ra* is viable, since even *Op* heterozygotes have reduced viability and *Ra* homozygotes rarely survive after birth.

Summary

A new hair deficiency of the mouse, called "Opossum," is due to an autosomal semidominant lethal mutation, *Op*, at a locus linked with the *a* locus in linkage group 5, with 26.8 percent of recombination in male coupling and repulsion heterozygotes. *Op* may be an allele of *Ra* (Ragged).

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BEHAVIOR GENETICS

THIS book* is an excellent one and greatly needed in a field of present emphasis and rapid development. The scope is defined in Chapter 1. The second chapter reviews some principles of genetics for those not familiar with the subject, and is followed by chapters on experimental methods in behavior genetics and methods employed in studies of human genetics. Then, in order, are chapters on variation in sensory and perceptual processes, the response processes, intellectual abilities, personality and temperament, mental disorders, and heredity and individual differences in behavior.

The authors begin by introducing the reader to the nature-nurture problem in which so much prejudice has clouded the truth. "Men are different, but man has not always been open-minded in seeking the source of these differences." They point out that since the 1920's fewer scientists can be classified as "hereditarians" or "environmentalists," and that the interplay of environment with hereditary factors is better understood. Traits can be classified as (1) hereditary if most of the variation within the population is associated with differences in genetic endowment, (2) non-hereditary if there is little or no genetically determined variance, and (3) clearly affected both by heredity and environment.

They point out a fact—misunderstood by many—that "behavior" *per se* is not inherited, but rather that which is inherited is a mechanism which when subjected to certain stimuli results in particular responses. Slightly different physical mechanisms due to different constellations of genes produce different physiologies, resulting in different psychosomatic behaviors. Motivation in this hybrid field comes more from an interest in behavior than in genetics, and its findings have application to the theory of evolution, to mental deficiency and psychiatric disorders of value in marriage counseling, as well as to animal breeding.

(*Continued on page* 243)

**Behavior Genetics*. John L. Fuller and W. Robert Thompson. John Wiley and Sons. New York. 396 pp. 1960. $8.95.

THE INHERITANCE OF TAILLESSNESS IN MANX CATS

NEIL B. TODD*

THE OCCURRENCE of a population of cats on the relatively isolated area which comprises the Isle of Man has attracted attention and fostered speculation for over a century because of the fact that a comparatively high percentage of these animals show anomalies of the tail which often result in a sinecaudal phenotype.

Anatomically the variability among Manx cats may briefly be described as from one to six caudal vertebrae showing fusion, asymmetry, and reduction in size from the corresponding elements in the normal cat[1,6,8,9]. The present study was initiated in an attempt to elucidate the genetic basis of the inheritance of taillessness in Manx cats and to shed light upon the origin of this mutant. A review of the literature made it evident that no resolution of these problems could be derived from the data and observations of former workers. However, one of the principal observations, the noting of the presence of certain tailless populations among Oriental cats, had led to the formulation of a hypothesis which would have Manx cats derived from some Asiatic source. As no unequivocal historical verification or refutation of this point could be found, a plan was devised by which it could be established through various genetic techniques that no biological relationship existed.

Materials and Methods

A limited breeding program was undertaken and additional data on crosses were gathered by questionnaire and interview from breeders of Manx cats to determine the Mendelian mechanism underlying the inheritance of taillessness in these animals. All tailless stock used in this project either originated directly from the Isle of Man or were the known descendants of such cats.

Breeding Data and Discussion

There is little question that the normal tailed condition breeds true in cats. The occasional report of tailless kittens resulting from the crossing of two known tailed cats may probably be attributed to a congenital deformity or perhaps to a mutation at the Manx or some other locus. No breeder questioned in the course of this study could give an instance of tailless kittens arising from the mating of tailed cats, one or both of which had a Manx parent or parents. This evidence justifies the assumption that the penetrance of taillessness is 100 percent.

On the assumption that the tailed condition was due to homozygosity, Manx female No. 1 was mated seven times to tailed cats with the results appearing in Table I. Apparently this animal is heterozygous for the Manx factor by virtue of the production of tailed young. In the crosses summarized in Table II, all of the Manx females (except No. 19) produced tailed progeny at some time and may likewise be assumed to be heterozygous at the Manx locus. Thus, with the exception of cross 19, in each instance noted above the cross made was heterozygote to homozygote ($M +^m \times +^m +^m$†) and Manx appears to be dominant. The total distribution of the progeny from these crosses (omitting 19) is 31 Manx to 27 tailed. This fits the expected 1:1 ratio by chi-square analysis with a

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†All other suitable symbols have been previously assigned to other factors in the cat.

probability of 50–70 percent (χ^2 = 0.276). The following discussion seems to be in order.

The sex ratio among the sexed individuals of 33 females to 23 males, and more particularly of 20 females to 11 males among the Manx offspring, is interesting, if not significant. The latter case fits the expected 1:1 ratio with a probability of approximately 10 percent (χ^2 = 2.60). This will be further discussed below. The average litter size for the above crosses is 3.15 ± .32.

The records of Anthony[2] and Kennel[6] of two tailless females mated with normal cats give a total result of 30 Manx to 22 tailed among the young. These figures do not differ significantly from those obtained in the present study; but it should be noted that the record of Kennel is not of a known Manx cat and there is no assurance that taillessness in this individual had the same genetic basis as it does in Manx cats. The only other record of consequence is that of a Dr. Wilson (quoted in Darwin[3]). "Dr. Wilson crossed a male Manx with common cats, and out of twenty-three kittens, 17 were destitute of tails; but when the female Manx was crossed by common cats all the kittens had tails, though they were generally short and imperfect." These data are not presented in such a way as to make analysis possible, but insofar as can be judged they do not gain confirmation from the present or other studies.

Data on the reciprocal cross of a tailed male to a Manx female are scant. Only two such crosses have been made in connection with this investigation, both with the male (No. 24) from the third litter of animal No. 1. One other cross was provided by a breeder. These data and those of Davenport[4] are compiled in Table III. The ratio of 15 Manx to 12 tailed fits the expected 1:1 ratio of heterozygote to homozygote with a probability of 80–90 percent (χ^2 = .33). From these crosses sex linkage is eliminated since Manx males occur in the F_1.

The average litter size for the above crosses is 4.50 ± .68.

In the crosses of Manx females to Manx males, the heterozygosity of all the animals except Nos. 30, 32, 45 to 48, 50 and 52 was demonstrated by the production of tailed kittens (Table IV). Of two of the exceptions, 30 and 32, at least one is a heterozygote since a mutual offspring, animal No. 31, produced tailed kittens when mated to animal No. 29. Since No. 30 was also mated to No. 29 and no tailed progeny were obtained, he may well be a homozygote. Over-all, animal No. 30 sired 12 kittens,

TABLE I. Progeny of animal No. 1

Cross	Dam	Sire	Manx ♀	Manx ♂	Tailed ♀	Tailed ♂
1	1	2	1	0	0	1
2	1	?2	0	0	1	1
3	1	3	1	1	1	1
4	1	?3	2	0	0	1
5	1	4	2	2	0	0
6	1	5	2	0	2	0
7	1	6	2	2	0	0
Total			15		8	

TABLE II. Results of crossing Manx ♀♀ with tailed ♂♂

Cross	Dam	Sire	Manx ♀	Manx ♂	Tailed ♀	Tailed ♂
8	7	8	2	0	2	2
9	9	10	0	0	3	0
10	11	12	0	0	1	1
11	11	12	2	1	0	0
12	11	12	2	1	0	0
13	11	12	0	1	0	0
14	11	12	1	0	0	0
15	13	14	0	1	2	2
16	13	15	1	0	0	0
17	13	16	1	1	1	0
18	17	18	1	0	0	2 + 2 unsexed
19	19	20	5	unsexed	0	0
20	21	22	0	1	0	1
Total			21		19	

TABLE III. Results of crossing Manx ♂♂ with tailed ♀♀

Cross	Dam	Sire	Manx ♀	Manx ♂	Tailed ♀	Tailed ♂
21	23	24	0	1	2	2
22	25	24	1	1	0	1
23	26	27	3	unsexed	3	unsexed
Davenport, 1905			3	2	1	0
			3	unsexed	1	0 + 1 unsexed
			0	1	1	0
Total			15		12	

all Manx, while No. 32 gave birth to nine Manx and no tailed kittens. No further information is available on Nos. 45 to 48, 50 and 52. On the basis of the above considerations, the statistical treatment of the data in Table IV, with respect to 3:1 and 2:1 ratios, includes only animal No. 32 among those for whom no proof of heterozygosity exists, although certain of these animals are doubtlessly heterozygotes. Therefore, the ratio taken for analysis is 63 Manx to 27 tailed which fits the expected 3:1 ratio of a heterozygote to heterozygote cross with a probability of approximately 30 percent (χ^2 = 1.20).

Once again the sex ratio among the Manx individuals, 47 females to 34 males, shows a preponderance of females. Statistically these figures fit the expected 1:1 ratio with a probability of about 10 percent (χ^2 = 2.086) and a 2:1 ratio with a similar probability (χ^2 = 2.715).

The average litter size for these crosses is 2.95 ± .20.

Table V gives data on the average litter sizes of the three crosses involving the Manx factor and that for normal or non-Manx matings, 3.88 ± .22 (from Hall and Pierce[5]). The correspondence of these crosses as determined by Student's t-test is summarized in Table VI. Taking the .05 level of significance, the crosses involving Manx females (*i.e.* Manx female × tailed male and Manx female × Manx male) show a 19 to 34 percent reduction in litter size which is significantly different at the .05 level, or nearly so, from those involving tailed females.

TABLE IV. Results of crossing Manx ♀♀ with Manx ♂♂

Cross	Dam	Sire	Manx ♀	Manx ♂	Tailed ♀	Tailed ♂
24	1	24	3	1	0	0
25	1	?28	0	1	1	1
26	1	28	0	1	0	0
27	29	30	2	1	0	0
28	29	31	0	0	0	1
29	29	31	1	0	0	1
30	29	31	1	1	0	0
31	32	30	1	1	0	0
32	32	30	0	1	0	0
33	32	30	2	1	0	0
34	32	30	1	2	0	0
35	33	34	1	1	0	2
36	33	34	0	2	0	2
37	33	34	1	2	0	0
38	33	34	1	2	0	0
39	35	36	1	1	0	1
40	35	37	1	1	1	0
41	35	38	0	1	2	1
42	35	38	1	1	0	1
43	35	39	1	1	0	3
44	35	40	1	2	1	0
45	41	42	2	1	0	1
46	41	38	2	0	1	0
47	43	42	0	2	1	0
48	43	38	2	0	1	0
49	43	40	2	1	1	0
50	44	40	4	2	0	0
51	44	40	2	unsexed	1	unsexed
52	44	38	4	unsexed	0	0
53	45	46	3	1	0	0
54	45	46	2	0	0	0
55	47	48	5	1	0	0
56	49	50	2	1	0	0
57	49	51	2	0	0	1
58	49	52	1	0	0	0
59	49	53	1	unsexed	0	0
60	54	55	0	0	1	0
61	54	55	1	0	0	0
62	54	55	0	1	0	1
Total				88		27

Conclusions

Any further explanation of the mechanism functioning in the inheritance of taillessness is

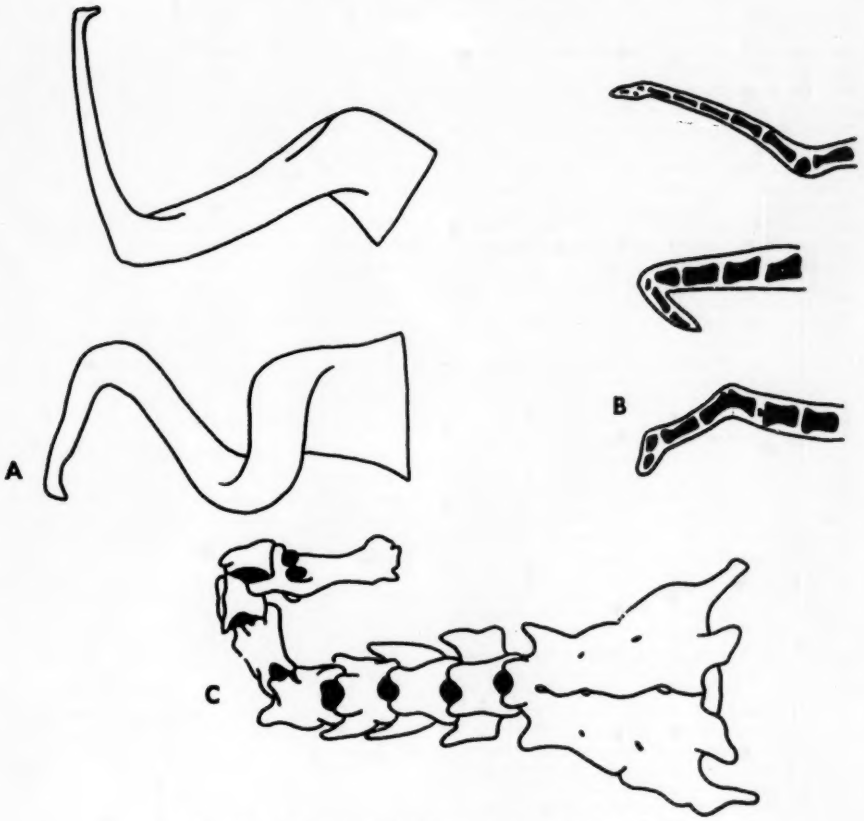
TABLE V. Summary of litter sizes for various crosses

Cross	Average	Source
Tailed × tailed	3.88 ± .22	Hall and Pierce (1934): 128 kittens/33 litters
Manx ♀ × tailed ♂	3.15 ± .32	Tables I and II: 63 kittens/20 litters
Manx ♂ × tailed ♀	4.50 ± .68	Table III: 27 kittens/6 litters
Manx × Manx	2.95 ± .20	Table IV: 115 kittens/39 litters

TABLE VI. Results of t-tests

Cross	T♀ × T♂	M♀ × T♂	T♀ × M♂	M♀ × M♂
T♀ × T♂	—	1.98	1.03	3.12
M♀ × T♂	1.98	—	1.96	.53
T♀ × M♂	1.03	1.96	—	2.71
M♀ × M♂	3.12	.53	2.71	—

t = .53, df = 59, p > .50; t = 1.03, df = 39, p > .20; t = 1.96, df = 53, p ≅ .05; t = 1.98, df = 26, p ≅ .05; t = 2.71, df = 45, p < .01; t = 3.12, df = 72, p < .01.



TYPICAL TAIL ANOMALIES IN ORIENTAL CATS

Figure 12

A—Skinned tails showing typical twisting and truncation; *B*—Alizarin transparencies of distal parts of kinky-tails, with abnormal vertebrae at the kinks; *C*—Skeleton of tail with severe hook-like kink, involving abnormalities to six caudal vertebrae; the sacrum is asymmetrical. (Camera lucida drawings from Searle, *Journal of Genetics*, 1960. Reprinted with permission.)

contingent upon the integration of several observations. Not the least important of these is the fact that breeders over a period of 50 years have failed to produce a "pure" strain of Manx cats. This immediately suggests a lethal function (pleiotropy) or factor (autosomal linkage) associated with the sinecaudal phenotype. This is not inconsistent with a second observation, *viz*, that virtually all Manx cats tested in the course of this study behaved as if heterozygous for taillessness, although the majority of these animals are the result of years of selection against such a condition. Finally, a preponderance of females among the progeny of Manx dams and a reduction in litter size in crosses involving Manx females have been noted.

The question arising directly from this information, and of the greatest significance, is that concerning the ratio obtained from the heterozygote to heterozygote cross. This ratio of 63 Manx to 27 tailed fits the expected 3:1 ratio with the probability noted above. However, these data also fit a 2:1 ratio with a probability of approximately 50 percent ($\chi^2 = .450$), so that lethality or partial lethality cannot be eliminated as a possible explanation of the apparent paucity of homozygous Manx cats. At the same time, the preponderance of females among the progeny resulting from crosses of Manx females by either Manx or tailed males might be due to a lethal function of the Manx allele which acts more detrimentally in the male than in the female. This latter suggestion would then contribute an explanation to account for

the reduction in litter size in matings of Manx females without the necessity of assuming that the maternal parent of such crosses manifests any reduced fertility. That is to say, the litter size is lowered due to a reduction in the number of Manx males which survive until parturition rather than any incapacity on the part of the dam to produce a normal sized litter. In fact, if the sex ratio among the Manx offspring of these crosses is restored to a 1:1 by increasing the number of males per litter to the corresponding number of females, the resulting average litter size accords closely with the normal or expected.

The most logical alternative which explains the apparent lack of homozygous Manx cats, if 100 percent lethality is eliminated as a possibility, is that cats of the genotype *MM* are sterile. This consideration has some support in the preceding evidence, although no direct tests of such a hypothesis have been attempted. Hence, if the discrepancy in the sex ratio of Manx kittens from the crosses involving a Manx female is assumed to be due to chance alone and the females are assumed to show a reduced fertility, an allele dosage effect can be postulated in which the heterozygous condition is less fertile than the homozygous recessive and the homozygous dominant is sterile.

Returning to the origin of the Manx cat and its biological relationship to other populations of tailless cats, the following information and work are noteworthy.

Oriental tailless cats differ phenotypically from the Manx cat in being semicaudal instead of more or less sinecaudal. Tails several inches in length but still much reduced from the normal exist in the former group, while in the latter such a condition is encountered only rarely. A further feature of the Oriental cats is the multiple kinking of the tail, which is occasionally seen in the Manx[7]. Crosses of Manx cats were made to compare the expression of the Manx factor in the Oriental genome. Imported and registered lines of Burmese and Siamese cats were utilized for this purpose since both breeds are derived originally from populations in which the incidence of the above described anomalies is common. The results of such crosses have consistently yielded, in the F_1 and F_2, progeny which with respect to taillessness conform to the Manx type, while such other characteristics as coat and eye color, eye, ear and body shape, voice, *etc.* are distinctly Burmese and/or Siamese. An additional distinction may be drawn between the two groups in that the taillessness of Oriental races appears not to have 100 percent penetrance, whatever the mode of inheritance[7], while it most surely does in Manx cats. From this evidence it is concluded that the caudal aberrations of the two groups being compared probably have two independent etiologies and are not due to a single factor working within two different genetic backgrounds.

Summary

Data gathered on crosses involving Manx cats, while not altogether statistically conclusive, lend support to the suspicion that taillessness is at least partially lethal. There is a statistically significant difference in the litter size produced by two Manx cats ($2.95 \pm .22$) and that produced by two tailed cats ($3.88 \pm .20$).

Attempts to place the Manx factor in an Oriental genome support the contention that taillessness in these two populations is due to two different mutants.

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INHERITANCE AND ORIGIN OF NECTARLESS MUSKMELON

A Yield-Reducing Mutant Character

GUY WESTON BOHN*

NECTARLESS (Figure 13), a new mutant character that renders muskmelon flowers unattractive to honey bees and thereby reduces yields, has previously been described[1]. The inheritance and origin of this character are discussed here.

Materials and Methods

Plants in various progenies related to those that exhibited the nectarless condition were grown to flowering from four, six, or eight seeds planted in marked positions in seven-inch clay or plastic pots in the greenhouses at La Jolla, California. Three to eight pots were used for a progeny row. Homozygous nectarless inbred progenies and the normal No. 45 and No. 450 varieties served as checks.

The plants were maintained in good condition by watering the soil daily with an excess of nutrient solution. Concentrated Hoagland and Arnon's[3] stock solution B, made to contain 10× the desired application rate with technical or fertilizer grade salts and acidified with 1000 p.p.m. of nitric acid, was diluted about one-tenth at each water application with a garden siphon inserted between the water faucet and hose.

Three or more flowers from each plant were scored for nectary size and condition between 10 a.m. and 2 p.m. Scoring errors resulted from (1) occasionally poor development of normal (nectar-producing) flowers, (2) the scoring of normal flowers too early in the day during dull, cool weather, (3) the accidental removal of nectar from normal nectaries by contact with dissecting tools or floral organs, (4) the visitation of normal flowers by bees in unscreened greenhouses, and (5) the production of small amounts of nectar by some flowers on genetically nectarless plants. Apparently nectary size and condition were affected by other genetic factors that had minor effects, because the listed errors occurred more frequently in some progenies than in others. However, the three readings obtained from a plant were usually identical in all progenies. The nectaries were affected also by the environment. Few errors occurred in the phenotypic identification of plants grown in a heated, screened greenhouse during sunny weather, so that the three sample flowers on a plant usually received a similar score. Nectary condition was variable in an unheated, unscreened greenhouse during cool, cloudy weather in winter, so that it was necessary to examine five to ten flowers on an occasional plant to establish its phenotype.

TABLE I. Segregation of plants with nectarless and normal nectaries in inbred muskmelon progenies

Cross	Generation	Normal (+/)	Nectarless (n/n)	Total	χ^2
n/n × +/+	F_2	382	140	522	0.92
+/n × +/+	F_2 segregating	71	23	94	0.01
+/n × +/+	F_3 segregating	233	73	306	0.21
	Total	686	236	922	0.18

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Sincere appreciation is extended to Thomas W. Whitaker, principal research geneticist, for valuable suggestions; to G. A. Sanderson, agricultural aid, for the aluminum, position-marking dibble and the photographs; to Joseph Principe, scientific aid, for the nutrient solution siphon-dilution technique and other technical assistance; and to Randolph Wedding, biochemist, University of California, Riverside, for advice on culture techniques.

NECTAR AND NECTARLESS FLOWERS

Figure 13

Staminate muskmelon flowers with parts removed to show the normal, saucer-shaped nectary with its drop of nectar at left and the smaller, columnar, nectarless nectary with minute droplets of nectar at right.

Segregation of Nectarless and Normal

F_2 progenies from the cross homozygous nectarless (dry) × normal (wet) [(P_3 × No. 45 × No. 450) F_3 × No. 450] and segregating F_2 and F_3 progenies inbred from the heterozygous trihybrid [(P_3 × No. 45) F_1 × No. 450]F_1 segregated 686 normal and 236 nectarless plants (Table I). That proportion, with a chi-square value of 0.18, presented convincing evidence that the nectarless character was controlled by a single, recessive, Mendelian factor.

The observation that all plants of numerous F_1 hybrid progenies (including those shown in Figure 14), as well as all plants of the No. 45 and No. 450 varieties, had normal, nectar-producing nectaries supported the single-factor hypothesis. It was substantiated also by the uniformly nectarless plants obtained in progenies derived from self-pollinated nectarless parents.

Observations on numerous other progenies grown in the field as well as in the greenhouse for use in the breeding program further supported the single-factor hypothesis. The symbol *n* is suggested to indicate the nectarless factor.

Origin of the Nectarless Mutant

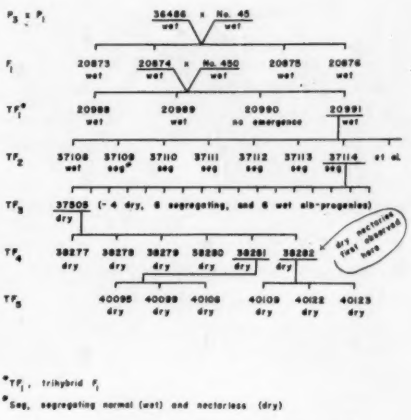
A survey of breeding stocks demonstrated that the trihybrid F_3 progeny 37505 and all of its F_4 and F_5 derivatives were homozygous for the nectarless factor (Figure 14). The character was not observed in any of the three parents, Nos. 45, 450, and P_3 progeny 36486. Similarly, the nectarless condition was not found in inbreds derived from the parents of P_3: PMR (powdery mildew resistant) selection 31015 from the cross No. 45 × RC (powdery mildew resistant inbred line designated Resistant Cantaloupe), and from U.S.P.I. 124111, a semi-feral muskmelon imported from India (Figure 15). No viable inbreds were available from parent 29362, a sib-line of 31015. These

observations indicated that the nectarless condition resulted from a new mutation that occurred in one of the individual parent plants used to make the crosses or in one of the early generation hybrids.

The genotypes of several individual plants listed in the pedigree in Figure 15 were determined by progeny tests. The genotypes of six individuals in the F_1 progeny 20874 were three $+/+$ and three $+/n$ (Figure 16). Obviously, one of the parents was heterozygous for the nectarless factor and the other was homozygous for the normal allele. Unfortunately, no selfed progenies were obtained from the two plants crossed to produce 20874. Either the pistillate parent plant 36486-2 or the pollen donor plant of the No. 45 variety could have been heterozygous for the nectarless factor. Hence, the nectarless mutant could have resulted from (1) a new or an old mutation in the No. 45 variety or (2) a new mutation in plant 36273-2. Evidence for the two hypotheses is largely circumstantial.

The nectarless character was not observed in examination of several dozen plants of the No. 45 variety. However, those plants were obtained directly from commercial seed without inbreeding. Whitaker and Bohn[5] and others demonstrated that considerable cross-pollination prevails in the muskmelon. Therefore, the nectarless mutant could have been carried as a hidden recessive in commercial seed stocks for an indeterminate period. Determining the presence or the absence of the nectarless gene in commercial seed stocks might require extensive self-pollination and flower examination. That procedure appeared to be impractical.

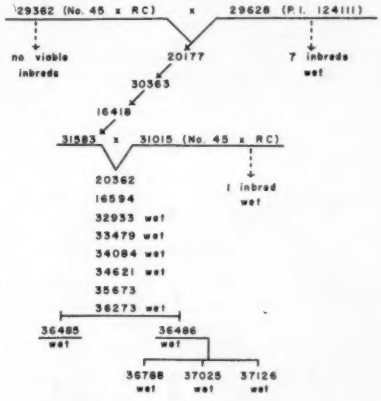
In contrast, the nectarless gene could not have been carried as a hidden recessive in the closely inbred, powdery mildew resistant parental line, for its occurrence there would have been detected in the P_3 progeny 36486 and its relatives. If the mutant originated in P_3, then it must have occurred as a somatic or other point mutation in plant 36486-2 or its parent 36273-2. Evidence from various sources suggests



FIRST NECTARLESS MELON

Figure 14

Pedigree of muskmelons in which the nectarless character was first observed.



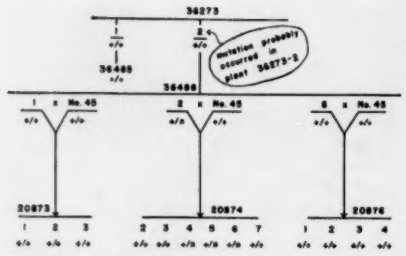
NORMAL NECTARY

Figure 15

Normal nectary condition in the pedigree of muskmelon P_3 (Powdery Mildew Resistant 36486).

that the mutation did, in fact, occur in one of those two plants.

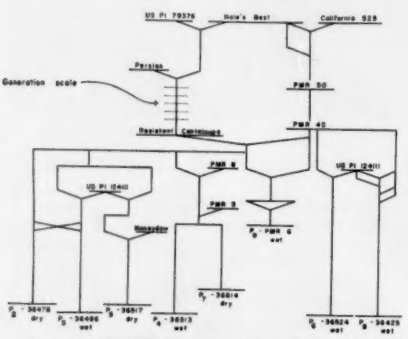
Convincing evidence that the nectarless gene originated in the resistant, rather than the susceptible, parent was found in the anomalous segregation observed in the trihybrid F_1 20991. Prog-



PROBABLE ORIGIN OF NECTARLESS

Figure 16

Genotypes of individual muskmelon plants showing the probable origin of the nectarless factor.



INBRED LINES RESISTANT TO POWDERY MILDEW

Figure 17

Pedigrees of eight inbred muskmelon lines selected for resistance to powdery mildew.

eny 37108, which was obtained from selfed 20991, was homozygous normal. All of its 12 sib-progenies tested were segregating normal and nectarless plants. The ratio of one homozygous normal to 12 heterozygous normal plants in 20991 is very significantly outside the range of the expected one to one ratio (chi square, 9.3). The apparent discrepancy is resolved if the nectarless factor was linked with a factor for resistance to powdery mildew and entered the cross from the resistant, P_3, parent. Such linkage would increase the numbers of genetically tested trihybrid F_1 plants heterozygous for nectarless because the 13 plants that were self-pollinated were selected for resistance to powdery mildew.

Additional evidence that the nectarless gene originated in the P_3 parental line was found in a study of the nectaries of other inbred lines of powdery mildew resistant muskmelons. The flowers of three of eight inbred lines, P_2, P_5 and P_7, were found to have dry nectaries (Figure 17). The defective nectaries of P_2 and P_5 resembled those found in P_3 derivatives; those of P_7 were small, round, and yellow-colored, differing in both structure and color from the others. The occurrence of nectarless flowers in four, including P_3, of six inbred lines of related parentage (from the cross Resistant Cantaloupe × No. 45) strongly suggests that they had similar origins and that all four originated in powdery mildew resistant inbred lines.

The occurrence of pollen sterility[2], yellow-green foliage[4], and several unreported mutants (including seed and seedling lethals in inbred lines derived from crosses between semi-feral powdery mildew resistant muskmelons imported from India and commercial varieties) supports the hypothesis that the nectarless mutant described herein did originate in the P_3 inbred line. The comparatively frequent occurrence of such defective mutants in inbred lines derived from such crosses suggests further that the mutants occurred following cross-overs between non-identical chromosomes differing in structure. The resulting chromatin deficiencies, duplications, and rearrangements would then produce sufficiently numerous defective individuals to account for those observed.

Summary

Nectarless muskmelons were found to differ from normal plants by a single, recessive, genetic factor. Pedigree records, linkage with factors for powdery mildew resistance, the occurrence of the same or similar characters in related inbred lines, and the common occurrence of other mutants in inbreds suggest that the mutation from normal to nectarless occurred in a single F_8 or F_9 plant of the powdery mildew resistant inbred line P_3 (36486). The frequent occurrence of defective mutants of various sorts in inbred lines derived from crosses between semi-feral powdery mildew resistant plant in-

troductions and commercial varieties of muskmelons suggests that those two groups of parents possessed chromosomes that differed in structure.

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◆

SNOW-WHITE DOWN IN DUCKS

JAROSLAV KRIZENECKY*

TEN years ago Hutt[1] described a mutation in the chick which caused the down, normally light or dark yellow, cream, or greenish white, to change to pure white, without affecting the yellow pigmentation of skin and eyes or pigmentation and barring in adult birds. Hutt named the mutation snow-white down. Black and red down remain unaffected by this gene. This snow-white is a unifactorial, autosomal recessive mutation.

On a commercial duck farm in Czechoslovakia, a snow-white down was found in day-old White Peking ducklings which parallels Hutt's mutation in chicks (Figure 18). Although it has been impossible to carry out the crosses necessary for exact evaluation of the genetic basis of this characteristic, a short description might be of interest.

The white color of hatched ducklings was of the same snow-white character as Hutt described in the chick. There were no differences between snow-white and yellow-downed ducklings in the yellow pigmentation of the shanks and bill, in the blue pigmentation of the eyes, or in adult birds. According to the records of the farm, no differences were found in viability and growth rate. Only hatchability decreased in snow-white as compared with the average hatchability in the normal yellow-downed flock, but this was caused by inbreeding, which will be discussed below. The average egg production of the snow-white-downed ducks was only a little below the farm average.

The first ducklings with snow-white down were observed in the spring of 1955 among ducklings hatched from eggs collected from January to April in a commercial flock of 350 ducks and 70 drakes. In this period about 20,000 ducklings from 33,000 eggs were hatched (hatchability about 60 percent). Four snow-white females and two snow-white males were hatched and reared. As these were not pedigreed hatches, the parents of these ducklings were unknown.

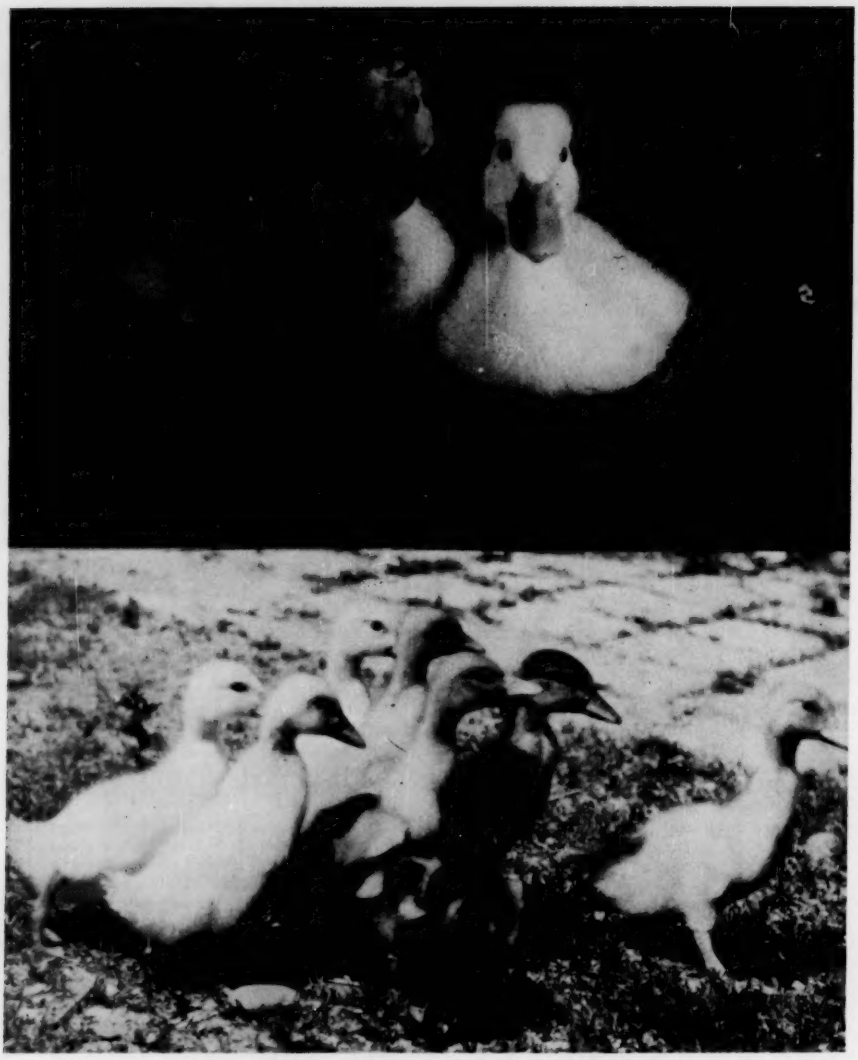
In the 1956 season one of the snow-white drakes was mated to four yellow-downed females hatched in 1955 in the same commercial flock (Pen A), and 240 ducklings were obtained, five of which were snow-white (sex not recorded). As these eggs were not pedigreed, the parents of the snow-whites could not be identified. The progeny of Pen A were not bred further.

A second snow-white-downed drake, hatched in 1955, was mated to the four snow-white females produced in 1955 (Pen B). Altogether 120 females and 90 drakes were obtained. All of them had snow-white down. (One of the snow-white mothers died during the breeding season.)

In 1957, three breeding pens were set up, two consisting of six ducks and one drake each of the snow-white birds hatched in 1956 and taken at random. The third pen was the same as Pen B of 1956. From these three pens 678 ducklings were hatched, all of which had snow-white down. The sex ratio was not recorded.

In the 1958 season, two new pens, each con-

* Brno, Susilova 11, Czechoslovakia.



SNOW-WHITE AND SIBLINGS

Figure 18

A normal White Peking duckling is shown with a snow-white mutant in the top photograph. In the lower photo the three ducks on the left are snow-white; the two next to them are White Peking. The two darker ones in the center are White Peking × Khaki Campbell.

sisting of six ducks and one drake from the snow-whites hatched in 1957 and taken at random, were used for breeding. From these two pens, 444 ducklings were hatched, all of which had snow-white down. Sexes were not recorded.

In the 1959 season two new pens were again set up, each consisting of six ducks and one drake taken at random from the snow-white stock. From these two pens, 1,045 ducklings were hatched, all of which had snow-white down.

It was possible to examine the snow-white

ducklings only once, late in the 1957 season, at which time a system of test-crossing was planned for 1958. However, circumstances prevented these experiments from taking place, and the administration of the farm, not being interested in research work, decided to discontinue further breeding experiments with snow-white ducks after hatchability fell to 31 percent in 1959. Accordingly, all the birds were killed.

The decrease of hatchability can be explained by inbreeding, as all members of the line originated from one drake and four female ducks, probably relatives, in Pen B of the 1956 breeding season. It is interesting to note that inbreeding did not affect egg production, as is seen in the summary of reproduction results on the farm (Table I).

As the snow-whites segregated originally from yellow-downed parents (both in the commercial flock and in the special mating of 1956) but when mated *inter se* produced only ducklings with snow-white down, it can be concluded that snow-white down in ducks is a recessive character. Whether it is unifactorial, a simple recessive, or is based on two duplicate genes cannot be decided because of the low percentage of snow-white ducklings from Pen A in 1956 (five birds against 235 yellow-downed). Even if it is assumed that these five birds originated from one heterozygous mother-duck, and calculated that she produced the average number of 60 ducklings (four female ducks produced 240 ducklings), the five snow-white birds represent only 12 percent of the progeny of this supposed heterozygous mother against the theoretical 50 percent expected from a backcross of unifactorial, homozygous recessive to heterozygote. Similarly, in the original commercial flock, the six snow-white ducklings, if produced by only one heterozygous mother-duck (and one heterozygous father), would represent only 10.6 percent of the average number of ducklings of one mother (the average number of ducklings in this case being 57, since 350 ducks hatched about 20,000 ducklings) against the theoretical 25 percent.

The relation of snow-white down to sex could not be determined, as the sex of the birds was not recorded.

TABLE I. Reproduction by snow-whites and other ducks

Year	Eggs per bird Average		Hatchability Percent	
	All ducks	Snow-whites	All ducks	Snow-whites
1956	—	122	—	61.7
1957	128	139	56.3	*
1958	166	135	50.4	*
1959	146	124	49.4	31.2
1960	131	—	58.4	—

* Not known.

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◆

A Preliminary Study of Inherited Blood Group Antigens in Families Having an Index Case of Early Infantile Autism

JACQUES MAY AND L. C. DUNN*

ASSOCIATION between a mental disease syndrome and some inherited blood group antigens has recently been tested. A primary difficulty in studies dealing with mental disease is that of definition. In determining the occurrence of "early infantile autism," which was selected for study in this instance, the following set of criteria was applied in each case:

1. The symptoms appear in infants and children under five; hence, little distortion is to be expected by unrelated symptoms appearing later in life.

2. Negative signs: The child should not show any indication of mongolism, deafness, brain tumor, hydrocephaly or neurological disease, defects of vision, etc.

3. Positive signs: (a) Speech disorders: either absence of speech or loss of established speech, or irrelevant speech (echolalia); (b) disorders of behavior characterized by difficulties in

* The Parents' School for Atypical Children, Chatham, Massachusetts, and Columbia University, New York. The authors wish to express their gratitude to those who made index cases available and to the families of the index cases who submitted themselves to investigation. Cooperating organizations and individuals bear no responsibility for the conduct of this family study.

relationships with adults and other children, in contrast to continuous relations with objects; (c) ritualistic preservation of habits once taken; (d) repetitive actions, such as rocking, spinning, head-banging; (e) aloneness and withdrawal.

All doubtful cases were excluded from this study on the assumption that in a preliminary test, such as the one proposed, it was safer to exclude a case which ought on several grounds to be classified as having the above syndrome than to include an exogenous patient.

Cases accepted as index cases were made available by the following physicians and clinics who were responsible for primary diagnoses: Metropolitan State Hospital, Boston, Massachusetts; Katherine Rice, M. D., Washington, D.C.; Parents' School for Atypical Children, Chatham, Massachusetts. Others came from a research program initiated by May.

Methods

Samples of blood were drawn by venepuncture from each index case and from the parents, and where possible from siblings of the index case. Anticoagulants (usually ACD or Alsever's solution) were used, and the blood was typed within three days. Saliva samples were taken from most of the cases.

Typing was done at three different laboratories. The first 16 families (from Boston) were typed at the Ortho Research Foundation, Raritan, New Jersey, through the courtesy of Dr. Philip Levine. Twenty-six families (from Washington and Baltimore) were typed in 1957 at the Institute for the Study of Human Variation by Dr. Ruggero Ceppellini and Filomena Innella. After the discontinuance of this Institute, all subsequent families were typed at Mount Sinai Hospital, New York, and the New York City Board of Health Rh-Laboratory through the courtesy of Dr. Richard Rosenfield, Natalie Gibbel and Gladys Haber.

The following properties were tested: ABO groups, usually including reverse typing of serum antibodies; Rh-type using sera anti-D, anti-C, anti-E, anti-c and anti-e; MNS types; Duffy using anti-Fy^a only. Most but not all samples were also tested for Kell, Lewis, P and secretor status, while a minor portion of the samples were typed for Jk^a.

Results

Sixty-eight families, each including one child diagnosed as having early infantile autism, were examined in whole (parents, patient and siblings) or in part (one parent with patient and/or siblings, or patient only). In one family the index case was a pair of monozygous twins, both affected.

The blood group distributions found were approximately those to be expected from random mating within a European population, except in the case of the Kell antigen. Here 13 Kell-positive parents were found out of 98 tested, using only those cases in which both parents were tested by anti-Kell serum. These were distributed as follows:

	Mating Type		Cases
1	Father K+	Mother K+	1
2	" K+	" K−	8
3	" K−	" K+	2
4	" K−	" K−	40

The inequality between mating types 2 and 3 is noteworthy, since under random mating without selection these two types should be equally frequent, regardless of the frequency of Kell-positive persons in the population. The inequality observed, that is, eight out of 10 of one mating type and two out of 10 of the other where equal numbers are expected, has a probability of about .055 for each inequality, that is, .11. This means that the inequality observed may occur by chance in 11 percent of similar samples from the same population, and hence does not provide statistical evidence for the association of the disease in the offspring with the Kell mating type of the parents.

Attention is called to this result because the authors believe that in the case of a mental disease such as this, in which causal sequences are unknown or controversial, hypotheses connecting biological factors in parents and offspring should be carefully examined, if only to establish exclusion of those hypotheses which can be tested by reliable criteria such as those offered by blood group serology.

In the authors' opinion an hypothesis of Kell-incompatibility should not be excluded on the basis of this first test with rather small numbers. The data should be extended by more tests of this sort, having in mind, for example, the comparison of clinical signs of autistics from Kell-positive fathers with those from Kell-negative fathers; and tests for antibody in the Kell-negative mothers of Kell-positive index cases. Finally, other forms or combinations of antigenic incompatibility between mother and fetus should be considered and tested for exclusion wherever possible.

HEREDITARY SPASTICITY IN MICE

C. K. CHAI*

SPASTIC MUTANT

Figure 19

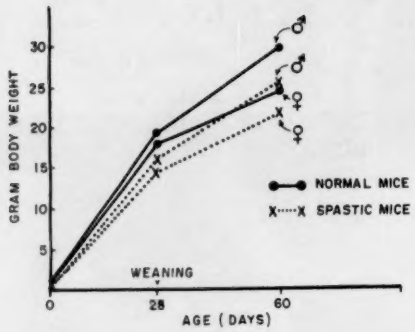
A mouse in the spastic condition shows the back stiff and arched, the legs stiff and stretched, and the toes extended.

ABOUT two years ago, a mouse with a neuromuscular disorder characterized by spasm was discovered in our random-bred stock, which had been developed by intercrossing six inbred strains[1]. After crossing this mouse with a normal mouse and intercrossing their offspring, individuals with the same syndrome were recovered. This preliminary breeding test suggested that the spastic condition behaves as an inherited character. A larger number of matings were set up to investigate the mode of inheritance. The results show that the inheritance of this condition is determined by a single autosomal recessive gene. In the present note we shall describe the syndrome shown by these mice and present the breeding data.

Description of Syndrome

Spastic mice may be recognized at about 14 days of age, but in some cases reliable classification cannot be made before five or six weeks of age. The spastic condition can be induced by sudden disturbance or handling. The period of spasm varies among animals. It lasts ordinarily from less than one minute to a few minutes. There appears to be a period of relaxation following the spasm during which no, or only relatively mild, spasm can be induced. The animals walk in short steps and on tip-toe. In the severe cases the animals are arch-backed and slide on the table (Figure 19). It is difficult to recognize the spastic animals when they are quiet in the cage, but when they are picked

*Roscoe B. Jackson Memorial Laboratory, Bar Harbor, Maine. This investigation was supported by grant C–3108 from the National Cancer Institute, National Institutes of Health, and In–19 from the American Cancer Society. The author is grateful to Joseph Morrison and Thomas Farley for their assistance in managing the animals and collecting the data, to Dr. W. T. West for histological examination of the muscles, and to Dr. E. D. Murphy for his interest and helpful discussion on the physiology of this mutant.



MEAN BODY WEIGHTS OF SPASTIC AND NORMAL

Figure 20

The mean body weights of spastic mice and their normal littermates at birth, weaning (28 days) and 60 days of age are illustrated graphically.

up by holding their tails, their backs become stiff and their bodies and legs quiver with a high frequency of vibration. The vibration can be felt by the hand holding the spastic. Sometimes the mice interlock their hind feet, and they have great difficulty in turning over without help after being laid on the table with the belly facing upward.

These mice lack facility in swimming.

A preliminary test was made on the spastic mice in response to drugs which affect the central nervous system. In a limited number of trials, some of the spastic mice showed slight improvement by injection of Diphenylhydantoin (Dilantin). Pregnancy seems to improve the condition, and in some cases to abolish it temporarily.

The percentage of mortality in the spastic mice is rather high before or around weaning age, although some of them can live almost a normal life span. The females can produce young but they are poor mothers, possibly because of physical difficulties which interfere with obtaining food, and with nursing. The males can mate, depending on the degree of spasticity. The frequency of litters from abnormal males is much lower than that from normal males. It was very difficult to obtain litters from matings of spastic females with spastic males.

Breeding Data

The results of the breeding test are given in Table I. The test includes three classes of matings, spastic × spastic, spastic × normal, and normal × normal. On the single recessive gene hypothesis, the observed ratios fit fairly well with the expected, except in the spastic × spastic class, where there was an animal with questionable classification. It was unfortunate that this mouse died not long after weaning and definite classification could not be made.

The birth, weaning, and 60-day body weights were taken for some of the spastic mice and their normal littermates. The spastic mice showed smaller body weights than the normal mice in the three different ages (Figure 20). Except for the birth weights of the females, the differences in the means are significant ($p < .01$).

No pathological changes have been found in the back and leg muscles of spastic mice. Histological studies of the central nervous system are being made. A preliminary sample showed no significant pathological changes.

TABLE I. Total numbers of spastic and normal mice produced from different types of matings

Matings		Offspring‡		
Type	*No.*	*Spastic*	*Normal*	*Unclassified*†
Spastic × spastic	2	15	1?	13
Spastic × normal*	11	54	68	32
Normal* × normal*	13	75	245	20

*The normal mice are produced by matings of normal × spastic.

†Dead before weaning; no reliable classification could be made; these are excluded from the analysis.

‡χ^2: In spastic × normal, for 1:1, $p>0.20$; in normal × normal, for 1:3, $p>0.50$.

Discussion and Conclusion

No definite conclusion can be made at present in regard to the location of the defects in spastic mice. In view of the fact that the severe spasm is not accompanied by pathological changes in striated muscle, the disturbance, if any, in the muscle must be functional. Whether there is any defect in the nervous system requires further histological and physiological examinations. The periodic tightening of the striated muscles in spastic mice seems to be somewhat similar to the conditions of myotonia congenita in goats[3], and Thomsen's disease in man. On the other hand, the positive response to an anticonvulsive drug suggests that a component of the functional disturbance in spastic mice may be similar to that in epilepsy. The most similar neuromuscular mutant in mice is Trembler[2], which is due to a single dominant gene. The young Tremblers show the symptoms of functional abnormality of spastic paralysis and convulsions, and the adults show a generalized tremor which ceases when they are at rest. Spastics are more viable than Tremblers.

From the breeding data it can be concluded that a single recessive gene is responsible for this condition. The symbol *spa* is proposed to designate the gene.

Summary

A spontaneous mutation causing spastic symptoms occurred in a random-bred stock of mice. Breeding tests indicate that this condition is hereditary and is due to a single autosomal recessive gene. The genetic symbol *spa* is proposed to designate the gene.

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Behavior Genetics

(*Continued from page* 227)

Some attempts have been made to use genetics as a research tool, which goes much farther than just working out the mode of inheritance of a trait. It proposes to synthesize behavior patterns. Behavior genetics has a contribution to make to education, psychiatry, clinical psychology and other professions dealing first-hand with a variety of human problems.

The review of the sensory and perceptual processes includes a discussion of temperature preference, taste threshold differences, olfaction, variations of the eye and visual perception, hereditary deafness, auditory discrimination, habitat preference and others.

The response processes are discussed under circling and choreic behavior, geotaxic orientation, the honey bee "sickle dance," autonomic responses, audiogenic seizures, specific courtship, movements, and many other items.

The discussion of intellectual abilities touches on familial correlation of intelligence, special abilities, twin studies, resemblance in I. Q., selection of intelligence in animals, psychological and physiological correlates of intelligence.

The Personality and Temperament section deals with the Bernreuter personality inventory to measure neuroticism, intraversion, dominance and self-sufficiency. Attitudes and interests show family correlations. On the animal side there is a discussion of learning ability, emotional and social behavior.

Under mental disorders the authors discuss schizophrenia, mania-depressus, involutional psychosis, neurotic disorders, criminality, homosexuality, mental deficiency, neurological defects, biochemical errors, epilepsy, etc.

In the last chapter are reviewed heritable variations in behavior in laboratory and wild populations, gene frequency of *Microcephalia vera*, race and class differences, the eugenics concept. These are followed by outlining the pathway between genes and behavior, enzyme paths, metabolic blocks, cholinesterase and adaptive learning, the pathway through hormones, and the pathway through the nervous system.

Various explanations, with diagrams, of the relation between genes, physiology and behavior are provided. Between genes and physiology the reviewer would have inserted the physical level at which variation occurs to determine altered physiology.

There is an extensive bibliography.

All persons interested in the behavior of man and the lower vertebrates will want a copy of this book.

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CRYPTORCHIDISM IN HEREFORD CATTLE

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CRYPTORCHID HEREFORD

Figure 21

First affected animal, D. He was the sire of B and the grandsire of C.

ARREST of testicular descent at any point along its regular path is known as cryptorchidism. One or both testicles may fail to descend into their normal positions in the scrotum as a result of developmental anomalies, mechanical obstruction or hormone deficiency. This sex-limited condition has been reported in man, horses, cattle, hogs, goats, dogs, cats, and other mammals.

Investigators have generally agreed that cryptorchidism is inherited. Lush *et al.*[3] reported there must be at least two pairs of recessive genes governing its occurrence in Angora goats, even if some cases are not truly hereditary. Their conclusions were later confirmed by Warwick[6] who worked with the same group of goats. McPhee and Buckley[4] concluded that cryptorchidism in swine is due primarily to the action of a single recessive gene and that unilateral and bilateral cryptorchids have the same genetic constitution.

This paper reports the occurrence of cryptorchidism in a purebred Hereford herd near Eastland, Texas.

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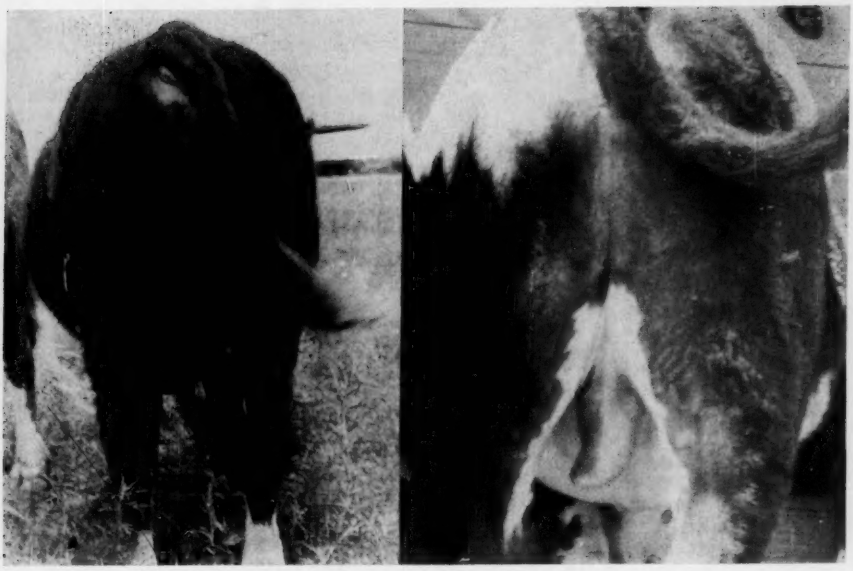


FATHER AND SON

Figure 22

Two affected animals showing different degrees of cryptorchidism. The bull on the left, B, was the sire of bull C, on the right.

Description of Affected Animals

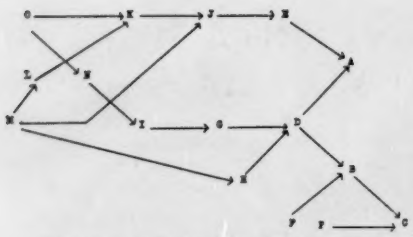
In a registered Hereford bull (animal D in the pedigree, Figure 21), the left testicle failed to descend into its normal position. This bull sired seven calves, two bulls and five heifers. One of the two male offspring (animal B in the pedigree, left in Figure 22) exemplified typical unilateral cryptorchidism, as his left testicle completely failed to descend into the scrotum. The writer did not see the other male offspring (animal A in the pedigree, Figure 23) but, according to its owner, the bull's left testicle failed to descend completely, evidently representing a condition midway between that of his sire, D, and normal.

Seven bull calves sired by B, from four to six months of age, were examined. By palpation it was determined that the left testicle of one calf (animal C in the pedigree, right in Figure 22) had descended to about the same degree as that of the left testicle of the calf's paternal grandsire, D.

Left cryptorchidism occurred in all four bulls. This may indicate ($P = .0625$) that in cases of cryptorchidism in cattle the left testicle is involved more often than the right, but the probability is not sufficiently small to draw a firm conclusion. Warwick[5] reported 36 cases of cryptorchidism in swine, three of which were bilateral. Among the 33 unilateral cases, the undescended testicle was the right one in eight cases and the left one in 25 cases. In 40 cases of unilateral cryptorchidism in Angora goats the right testicle, without exception, was the one which failed to descend (Lush *et al.*[3]). Hobday[2] reported 77 cases of cryptorchidism in horses; 11 were bilateral, 36 had the right testicle retained in the body, and 30, the left.

Nature of Inheritance

Cryptorchidism in these Herefords was first believed to be caused by a homozygous recessive condition. This



RELATIONSHIP OF AFFECTED ANIMALS

Figure 23

Pedigrees of four unilateral cryptorchid Hereford bulls, A, B, C, and D.

seemed to be the realistic approach as most of the presently known hereditary anomalies of domestic animals result from recessive genes. Also, though the writer found no description of inheritance of cryptorchidism in cattle (other than Gilmore[1] who reported that evidence indicates the condition to be inherited and that a recessive gene may be involved), reports of its inheritance in swine (McPhee and Buckley[4]) and goats (Lush *et al.*[3] and Warwick[6]) indicated recessive genes to be involved. If cryptorchidism in cattle is caused by recessive genes, inbreeding would increase its frequency. However, inbreeding could not have been a major factor in these cases as A was the only affected bull which was inbred and his inbreeding coefficient was only 4.3 percent (Figure 23).

Available evidence suggests an inherited basis for cryptorchidism in these Herefords, but is inadequate to allow the mode of inheritance to be determined. Thus, the hypothesis that it is caused by recessive genes, as in goats and swine, cannot be eliminated; but since relationships between parents of affected animals were so low, ranging from zero to only 8.6 percent, it seems that cryptorchidism in these Herefords also could have been caused by a dominant gene with variable expressivity.

Summary

Four cases of unilateral left cryptorchidism in purebred Hereford bulls are described. Pictures of three of the bulls are included.

Available information indicates an inherited basis for cryptorchidism in these Herefords but is inadequate to permit the mode of inheritance to be determined. The anomaly could have resulted from recessive gene action but a slightly preferred hypothesis is that it was caused by a dominant gene with variable expressivity.

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EYE-COLOR MUTATIONS FROM MUTANT STOCKS IN MORMONIELLA

P. W. WHITING*

THE majority of irradiation-induced eye-color mutations in *Mormoniella* are from wild-type brown stock. Relatively few have been obtained by treatment of mutant stocks because little of this work has been done. The present paper surveys these results with respect to the genic nature of parents and mutant offspring without reference to dose and rate, which would in these cases have little significance. Genes characterizing all parental stocks were X-ray induced, as were all the new mutant genes, except pellucid-311 and black-425.

The *R* locus is complex, containing many different factors which influence development, affect eye color, body size, viability, fertility and egg production. Strength of linkage of this locus with other loci in its vicinity is also affected. A mutation affects the state or condition of one or more of the factors, producing a new allele in the *R* series. Formula of a gene is expressed with its factor states arranged in an arbitrary order because within the gene there is no crossing-over, the whole complex segregating as a unit at meiosis. The three eye-color factors with which this paper is concerned are *O*, *S*, and *M*. The states of factors affecting fertility, viability, etc. may be given following the eye-color factors. Symbols expressing the factor states are separated (or rather tied together) by periods. The normal wild-type gene is +.+.+. Unstable-*O* *R*-locus spontaneous mutations from one mutant eye color to another are not considered here. They have been discussed elsewhere[4].

Table I lists the designations of the *R*-locus parental genes and of each mutant gene derived from the various parents. The gene formula in terms of factor states follows each designation. The six mutant genes given first are unifactorial, differing from the parental wild-type gene in a single factor. Other genes may be bifactorial or trifactorial. The genes are listed in Table I in the order in which they are discussed in the following text.

A dark red, dahlia-GF, is *da*.+.+, being a change in factor *O*. Another dark red, dahlia-846, is +.+.*da*, being a change in factor *M*. Factor *O* most frequently mutates from wild-type to oyster white, *oy*.+.+, as in oyster-DR, and factor *S* to scarlet, +.*st*.+, as in scarlet-DR. Mahogany-605, +.*mh*.+, and vermilion-MK, +.*vm*.+, resulted from changes in *S*. Mahogany is a darker red than dahlia, and vermilion is bright red with a dark fleck which is much more pronounced than the small fleck in scarlet.

X-irradiation of dahlia-GF females, *da*.+.+, produced a scarlet mutant, scarlet-426, which proved to be *da.st*.+, a change in *S* with the *da* condition of *O* unaffected. In another experiment with dahlia-GF, a pale-eyed mutant, apricot-837, *ap*.+ +, was obtained by a change from *da* to *ap* in factor *O*.

Treatment of dahlia-846, +.+.*da*, females produced an orange-eyed mutant, orange-806, +.*st.da*, and two oyster whites, oyster-825 and oyster-826, both *oy*.+.*da* as breeding tests showed. It is of interest that *da.st*.+ is brilliant scarlet in phenotype, as in scarlet-426, while +.*st.da* is very much lighter and duller, orange-806. The symbol *da* implies no chemical similarity between *da*.+.+ and +.+.*da* but only a resemblance in color between the two stocks. One oyster white mutant, oyster-848, *oy.mh*.+, was ob-

*Zoology Laboratory, The University of Pennsylvania. This work was done under Contract No. AT(30-1)-1471 between the U. S. Atomic Energy Commission and The University of Pennsylvania. Part of the project was carried out at the Marine Biological Laboratory, Woods Hole, Massachusetts.

tained from X-irradiation of a mahogany-605, +.*mh*.+, female; and three oyster white mutants, oyster-847, oyster-847.2 and oyster-847.3, all of gene formula *oy.vm*.+, were obtained from treating vermilion-MK, +.*vm*.+, females.

Like the oyster white mutants mentioned above, several oyster mutants have been obtained from X-irradiation of scarlet-DR, +.*st*.+, females without disturbing the status in factors *S* and *M*. Thus, oyster-422 and oyster-423 proved to be *oy.st*.+. The latter gene, however, carries a recessive for female sterility, *fsa*, so that the gene formula is *oy.st*.+.*fsa*. The homozygous females are sterile but the males are highly fertile.

Results were reported from a genetics class laboratory exercise in which wild-type and scarlet-DR females were X-rayed and the eye color of their sons scored[3]. Of 22 oyster-white mutant sons of scarlet mothers tested, 19 proved recessive to scarlet. These, oyster (19) in Table I, may be regarded as *oy.st*.+. The remaining three, oyster (3), were reported as complementary, producing wild-type compound daughters, +.*st*.+/*oy*.+.+, from scarlet mothers. These then would be, in a sense, double mutants, a "loss" in *O* and a "gain" in *S* from the +.*st*.+ gene. The occurrence of eight wild-type mutants, wild-type (8), from scarlet mothers is a "gain" in *S*. These tests have not been adequately checked and repeated, but pure stocks from several of the recessive oyster, oyster-NH (several), were derived and were shown to act as simple recessives to wild-type without scarlet appearing in F_2. They were, therefore, *oy.st*.+, not involving a locus other than *R*.

X-irradiation of females of one of these *oy.st*.+ stocks, oyster-NH1, resulted in a scarlet mutant son, scarlet-428. Complementation tests of scarlet-428 stock showed the gene to be *da.st*.+, similar to scarlet-426 mentioned above. Scarlet-426 was derived as an *S* mutation from *da*.+.+ and scarlet-428 was derived as an *O* mutation from *oy.st*.+. The *st* status of *S* prevents the formation of dark pigment, but the cross with complementary oyster, *oy*.+.+, masks scarlet, producing dark red compound females, *da.st*.+/*oy*.+.+. Similar complementation tests have been used in determining all the gene formulae given above.

White-eyed mutants acting as recessives to scarlet may be due to a change at some locus other than *R*. A few non-*R* whites have been found, and these, if they are derived from scarlet stock, produce scarlet among F_2 males from crosses with wild-type. Thus, in the derivation of pellucid-311, a spontaneous white often showing a very pale pinkish tinge, the F_2 males were 423 wild-type, 365 scarlet and 783 white, indicating very slight if any linkage.

An X-irradiated dahlia-GF female produced a lavender-eyed mutant male. This proved to be due to a mutation at a non-*R* locus, black-424. Black removes all the red color from the wild-type eye and with dahlia removing much of the black, the double recessive, dahlia black, should be dilute black.

TABLE I. Designations of *R*-locus genes and their formulae in terms of factor states

Parental genes		Genes of mutants	
Designations	Factors *O. S. M.*	Designations	Factors *O. S. M.*
Wild type	+. +. +	Dahlia-GF	*da*. +. +
"	+. +. +	Dahlia-846	+. +. *da*
"	+. +. +	Oyster-DR	*oy*. +. +
"	+. +. +	Scarlet-DR	+. *st*. +
"	+. +. +	Mahogany-605	+. *mh*. +
"	+. +. +	Vermilion-MK	+. *vm*. +
Dahlia-GF	*da*. +. +	Scarlet-426	*da*. *st*. +
"	*da*. +. +	Apricot-837	*ap*. +. +
Dahlia-846	+. +. *da*	Orange-806	+. *st*. *da*
"	+. +. *da*	Oyster-825	*oy*. +. *da*
"	+. +. *da*	Oyster-826	*oy*. +. *da*
Mahogany-605	+. *mh*. +	Oyster-848	*oy*. *mh*. +
Vermilion-MK	+. *vm*. +	Oyster-847	*oy*. *vm*. +
"	+. *vm*. +	Oyster-847.2	*oy*. *vm*. +
"	+. *vm*. +	Oyster-847.3	*oy*. *vm*. +
Scarlet-DR	+. *st*. +	Oyster-422	*oy*. *st*. +
"	+. *st*. +	Oyster-423	*oy*. *st*. +. *fsa*
"	+. *st*. +	Oyster (19)	*oy*. *st*. + (?)
"	+. *st*. +	Oyster (3)	*oy*. +. +
"	+. *st*. +	Wild type (8)	+. +. +
"	+. *st*. +	Oyster-NH (several)	*oy*. *st*. +
Oyster-NH1	*oy*. *st*. +	Scarlet-428	*da*. *st*. +

The bluish or lavender appearance is probably a structural effect as in the feathers of the Blue Andalusian fowl. This black in combination with scarlet-DR, from which it segregates independently, produces a rather opaque white as if there were white pigment present in the eye which is lacking in the translucent oyster. Scarlet black is white because *st* removes black pigment, *bk* removes red from the wild-type brown color. Some mutations from scarlet stock to white are, therefore, from another viewpoint, mutations to black.

A similar mutation has recently occurred spontaneously in scarlet-DR stock. Several rather opaque white males appeared, indicating that the mutation took place in an earlier generation. A pure-breeding white stock was derived and white females were crossed with wild-type males. Unmated wild-type daughters produced males—141 wild type, 132 black, 163 scarlet, 170 white. Daughters mated to their white brothers produced the four colors in both sexes. There were 179 scarlet, 175 white, 348 wild-type and black not separated. Black is distinct from wild-type in the males but difficult to separate in the females in which wild-type brown is very dark. It is clear that this black, called black-425, segregates, like the black-424, independently of *R*-locus scarlet.

Discussion and Summary

Somewhat over two dozen *R*-locus eye-color mutants are reported following X-irradiation of mutant stocks. The unifactorial genes of the irradiated stocks, differing from wild-type in a single factor, were thus, for the most part, changed to bifactorial genes, the majority of which mutated to *oy*, oyster white, in factor *O*, leaving the status of the other factors unaffected.

The *st*, scarlet, status of factor *S* was changed to +, the wild-type status, in 11 mutants reported by a student laboratory class. Three of these + mutants in *S* involved a simultaneous mutation to *oy* in *O*. While this student work should be checked and repeated, the results are not at all improbable. A "gain" in factor *O* from low to high color potency, even to +, is not infrequent in the unstable-*O* stocks[4] and simultaneous mutations in different factors have been shown to be 10 times as frequent as random expectation[2].

The eye-color factors are probably cistrons[1]. The numerous other *R*-locus factors are revealed by deleterious mutant changes probably caused by inversions, deletions, etc. associated with the alleles.

Recessive mutations at other loci affecting *R*-locus eye colors are reported.

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FACTORS AFFECTING SPOTTING IN HOLSTEIN-FRIESIAN CATTLE

II. Association of Head Pattern and Amount of Pigmented Body Area

J. M. TREECE, L. O. GILMORE AND N. S. FECHHEIMER*

LITTLE is known about the specific effects of inheritance on coat color and color patterns in cattle, although techniques are available to delineate certain gene effects rather sharply. Both color and pattern are used as criteria for registration in some of the herd books and both are used as identifying trademarks of many breeds of livestock.

Dunn *et al.*[3] suggested that a single pair of modifiers, quantitative in action, is largely responsible for varying amounts of unpigmented areas in Holsteins.

In studies involving spotted cattle (*ss*), Lauprecht[10] went further than Kroning[9] in postulating certain specific pigmentation centers, with a definite order of appearance of these centers in animals ranked according to progressively greater pigmented areas. Accepting this conclusion, Funkquist[4] also suggested independent factors for the inheritance of each center. Ibsen[7] postulated the occurrence of six specific genes modifying the expression of white spotting (*ss*). Briquet and Lush[2] have concluded that at least four pairs of factors are involved in the inheritance of spotting patterns.

In spotted breeds of cattle, it is generally agreed that there are five head patterns, although certain additional patterns have been cited by Funkquist and Boman[5]. These patterns are:

Whitehead. The head is entirely unpigmented with the possible exception of pigment around the eyes. The inheritance has been indicated by several investigators[6,8].

Blaze. The head has a strip of unpigmented area extending over the forehead and face from the region of the poll to the muzzle and varying in width.

Shield and Snip. There is an unpigmented area on the forehead which varies in size, with a smaller spot, the snip, usually on or near the muzzle.

Shield. There is an unpigmented area of varying size on the forehead.

Pigmented Head. The head is entirely pigmented. Funkquist and Boman concluded from their studies on head patterns in black-spotted Swedish lowland cattle and a red-spotted Swedish breed that four factors or pairs of genes were responsible, with multiple gene effects.

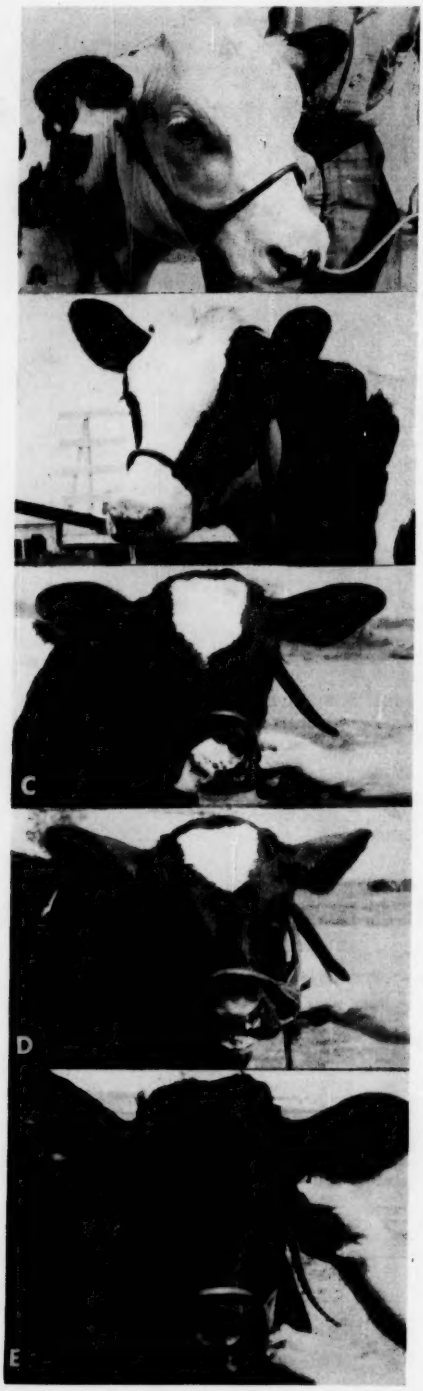
Kroning[9] found a high degree of relationship between members of identical twin pairs but, nevertheless, considered it to be improbable that the different markings, or pigmentation areas, are determined by specific genes. Lauprecht[10] stated that there was a positive correlation between pigmenta-

TABLE I. The mean amount and range of pigment area on the body for animals in each of five head pattern groups

Head pattern group	No. animals in group	Mean ± S.E.*	Range
		%	%
Whitehead	132	20.6±1.7	0–86
Blaze	316	41.0±1.1	2–89
Shield and snip	195	66.2±0.4	15–92
Shield	243	80.8±0.9	18–96
Pigmented head	15	89.8±1.3	79–97

*Differences among means are statistically highly significant ($P<0.001$).

*Ohio Agricultural Experiment Station, Wooster. Journal Article 84–59 of the Ohio Agricultural Experiment Station.



tion of the head, throat and legs and the total amount of spotted area.

Funkquist[4] concluded that the head patterns were not dependent for their expression on genes primarily affecting the rest of the body and that the head patterns constituted a separate hereditary complex.

In a study on the facial markings of Arabian horses, Blunn and Howell[1] found that the star, strip (blaze), snip and chin spot were conditioned by modifying factors of a general nature for body markings. The star, strip, and snip patterns were concluded to be inherited as dominant characters, while the chin spot appeared to be a recessive.

The data reported here on the relationship between head pattern and amount of body pigment in spotted cattle were part of a larger study dealing with the expression of spotting in Holstein-Friesian cattle.

Procedure

Data for this study were obtained from the same Holstein-Friesian herds as those reported previously[12], as was the method of visually estimating amount of pigmented area. More data were included than in the previous paper because of subsequent new additions to the herds and also because there was no elimination of data necessary for statistical analysis. Besides classifying the cattle as to amount of pigmented body area, they were also classified as to head pattern (Figure 24). Observations were made on 901 head of cattle sired by 93 bulls.

Results and Discussion

The data in Table I show the mean values for the amount of pigmented body area arranged according to head pattern. The differences among the mean values for each of the head patterns were statistically highly significant ($P < 0.001$)[11]. The range of values for the

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SPOTTED PATTERNS

Figure 24

The typical head patterns found in spotted cattle are shown on the left. *A*—White head; *B*—Blaze; *C*—Shield and snip; *D*—Shield; and *E*—Pigmented.

pigmented area shown within the head pattern groups and the regularity of differences of approximately 20 percentage units between the means of the first four head pattern groups suggests an association of head pattern and amount of body pigment. To check this possible association the cattle were placed into 10 groups each arbitrarily representing 10 percentage units on a scale of 100. The frequency as well as the actual number of cattle in each subgroup is shown in Table II. The over-all frequencies of the head patterns were .146, .351, .216, .270 and .017, respectively, for whitehead, blaze, shield and snip, shield, and pigmented head. This sample is considered to contain but little bias caused by excessive progeny by one bull or because of a high degree of relationship among bulls. Only one of the 93 bulls was used in two of the three herds and there were only a few cases of sire-son or brother combinations used in any of the herds.

The data shown in Table II indicate that there is a definite association between head pattern in spotted cattle and amount of pigmented body area. This suggests the existence of five head pattern genotypes with major effects upon body pigmentation. The variation within head pattern groups suggests the presence of several genes with minor effects that contribute to the within-head pattern variation.

Summary

It was found that there was an association of head pattern and amount of pigmented area on the body. The mean values of pigmented area in percentage units on the body associated with each head pattern were as follows: whitehead, 21; blaze, 41; shield and snip, 66; shield, 81; pigmented head, 90. It was concluded that the five genotypes which were responsible for head patterns were also largely responsible for controlling the amount of pigment on the body.

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TABLE II. Cattle grouped according to head pattern and amount of pigmented body area to show association of head pattern with amount of pigmented body area

Amount of pigmented body area	Whitehead		Blaze		Shield & snip		Shield		Pigmented head		Totals	
%	*No.*	%	*No.*	%	*No.*	%	*No.*	%	*No.*	%	*No.*	%
0– 9	42	4.7	11	1.2	0	0.0	0	0.0	0	0.0	53	5.9
10–19	42	4.7	36	4.0	3	0.3	1	0.1	0	0.0	82	9.1
20–29	19	2.1	63	7.0	4	0.4	3	0.3	0	0.0	89	9.9
30–39	13	1.4	48	5.3	10	1.1	2	0.2	0	0.0	73	8.1
40–49	4	0.4	54	6.0	20	2.2	5	0.6	0	0.0	83	9.2
50–59	3	0.3	38	4.2	27	3.0	8	0.9	0	0.0	76	8.4
60–69	3	0.3	29	3.2	28	3.1	14	1.6	0	0.0	74	8.2
70–79	4	0.4	29	3.2	54	6.0	36	4.0	1	0.1	124	13.8
80–89	2	0.2	8	0.9	42	4.7	116	12.9	6	0.7	174	19.3
90–100	0	0.0	0	0.0	7	0.8	58	6.4	8	0.9	73	8.1
Totals	132	14.6	316	35.1	195	21.6	243	27.0	15	1.7	901	100.0

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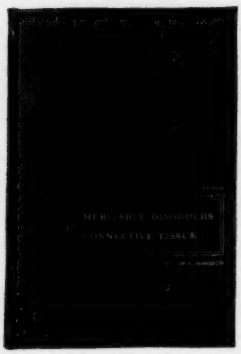

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